Discussion on the varieties and treatment of asthma / by G. A. Gibson.

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DISCUSSION ON THE VARIETIES AND TREATMENT OF ASTHMA.

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OPENING PAPER.

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ITS dramatic symptoms, its elusive pathology, its uncertain therapeutics may well be said to invest the subject of asthma with a cloud of mystery. As the mind of man has at all times delighted in attempts to look behind the veil which screens the unknown, there can be little wonder that the affection has exercised a fascination for the medical inquirer, and no more interesting topic could have been chosen for a discussion in this Section. It would obviously be out of place on such an occasion to submit a treatise on asthma. It will only be possible for me to lay before you some of the principal points connected with those particular aspects of the subject with which you have asked me to deal. But, inasmuch as certain of these matters cannot be effectively grasped without clear conceptions of the physiological and pathological foundations on which our present knowledge and beliefs are built, it will be necessary for me, with your permission, to spend two or three minutes on a few introductory considerations of this nature.

The facts which furnish a physiological basis for the classification of the causes of asthma naturally fall into two groups. There are, first, certain mechanical arrangements for the aëration of the blood. The upper air passages are obviously intended to warm and purify the air as it is inspired, and these regions are highly vascular, containing at the same time, in association with the blood supply, a considerable amount of erectile tissue. There is, therefore, in the arrangement of the structures, what may be looked upon as a defensive or protective mechanism. The lower air passages form practically two distinct categories —the larger tubes, with almost rigid walls, containing very little muscular tissue, and the smaller tubes, endowed with a considerable degree of contractility, and possessed

of a large amount of muscular tissue. An interesting point with reference to the smaller tubes is that the muscular tissue appears to have no tonus. One structural consideration must be brought before you in this connexion. As is well known, the cilia, which are found throughout a great part of the respiratory passages, do not penetrate into the smallest bronchial tubes. Some years ago, in an article contributed by the late Professor of Medicine in the University of Edinburgh and myself to the *Twentieth Century Practice* the opinion was expressed that the main reason why capillary bronchitis was so much more severe than bronchitis of the larger tubes lay in the absence of cilia in the smallest tubes. This is the first opportunity which has been afforded me of publicly referring to the subject, and it is only right, in justice to the memory of the late Sir Thomas Grainger Stewart, to state that the idea was his, and not mine. It is an idea which has sometimes been dealt with by subsequent writers, and it is a pleasure to me to show where the credit of the suggestion ought to lie.

The nervous arrangements subserving the respiratory movements not merely involve afferent tracts, regulating centres and efferent tracts, but also the control of all those arrangements by higher centres. It may be said that practically every sensory nerve in the body may carry influences acting upon the respiratory centres, whether of inspiration or of expiration. The efferent tracts form likewise a widely-spread ramification, although they are probably not quite so universally distributed as is the case with the centripetal fibres. We may take it, nevertheless, that the fifth, seventh, ninth, tenth, eleventh, and twelfth cranial, as well as a large number of the spinal nerves, are concerned in the efferent impulses connected with respiration. All these nervous mechanisms are controlled by higher cerebral centres.

As bearing most particularly upon the subject of asthma, the investigations of Brodie and Dixon are of the highest value. These observers, by their ingenious method of studying the volume of air entering and leaving the lung, when placed in the oncometer, have shown that the bronchial tubes are supplied by certain nerves which act as dilators, and by others which act as constrictors. Their physiological results are of the greatest importance for the apprehension of asthma. Some years ago Watson Williams suggested that during ordinary respiration there was probably a rhythmical dilatation and contraction of the bronchial muscular coat, in association with the movements of the nose, throat, and larynx. It seems to me that this is not merely a reasonable but a probable suggestion; and the researches of Brodie and Dixon have provided a reasonable foundation for it.

Such are the preliminary considerations which have seemed to me absolutely necessary to recall before we are in a position to deal with the subject of asthma.

Many theories have been suggested to account for the characteristic paroxysm of asthma. Floyer appears to

have been the first to advance the conception that the cause must be sought in a spasm of the bronchial tubes. This view was adopted by Cullen and Laënnec. After the discovery of the circular fibres of the muscular layer of the bronchial tubes, a contraction of these fibres was suggested by Williams and other writers as the real cause of the paroxysm. On the other hand, Walshe asserted that, at least in certain cases, a paroxysm of the bronchial muscular fibres, inducing a loss of expiratory power, was the more probable cause. A spasm of the diaphragm was suggested by Wintrich and supported by Traube, while a spasm of other inspiratory muscles was advocated by Budd, Jaccoud, and Germain Sée. A vasomotor neurosis, leading to hyperaemia of the mucous membrane of the bronchial tubes, was the opinion advanced by Weber and afterwards modified by Clark, while a somewhat analogous theory, invoking a spasm of the arterioles with, at the same time, an oedematous condition of the bronchial mucous membrane, was brought forward by Glasgow. Lastly, an acute catarrh of the mucous membrane was the view of Bree and Beau, while a specific inflammatory condition of the mucous membrane by microbic influences was suggested by Berkart.

The first part of the subject which you have asked me to bring before you deals with the *varieties of asthma*. From my own personal point of view this is an easy matter. To my way of thinking there is one form of true asthma—brought about, no doubt, by a number of different factors—of which there are a number of counterfeits. When turning this subject over in my mind in order to be ready to present it to you, a letter reached me from my friend, the Regius Professor of Physic at Cambridge. As it so entirely coincides with my own views, you will perhaps allow me to read it to you.

St. Radegund's, Cambridge,
July 11th, 1911.

Dear Gibson,—I fear I shall be unable to attend the British Medical Association meeting at Birmingham this month. I see you are going to open the discussion on asthma. You are careful about language, and I hope you will protest against the shifting of labels, which encourages loose speaking and thinking in students and in *others*. Thus the name *asthma* is often used for what is *not* asthma; for example, cardiac dyspnoea and uraemic dyspnoea; as slovenly people talk of uraemic or Jacksonian epilepsy, meaning convulsions only. These ways cause no end of clinical fog and react unfavourably on precision of clinical thinking. In a drawing-room a lady may call a whale a fish; but to see it so called in the technical papers of a zoologist would shock us; it is, however, no worse than *cardiac asthma*.—Yours always,

CLIFFORD ALLBUTT.

There is undoubtedly a real distinction between primary asthma and secondary dyspnoea. Frankly speaking, it is sometimes rather difficult in certain cases to determine whether an attack belongs to the former or to the latter category; but, as a rule, the distinction is not difficult to make, and the absence of the accompaniments of

secondary or symptomatic dyspnoea often clearly establishes the difference.

True neurotic asthma may be induced by a large series of causes. These factors may sometimes belong inherently to the nervous system. It occasionally happens that absolutely no cause can be elicited, and that the attack appears to take its origin in obscure influences affecting the central nervous system. Many of these cases manifest a rhythm or periodicity, as if some chemical material were stored up in the nervous centres and exploded at the end of a latent period. It is possible that modern theories of anaphylaxis may, in the future, aid us in our understanding of the reasons which lead to the perpetuation of such a rhythm.

More easy to grasp are those cases in which attacks are precipitated directly by the influence of the emotional and imaginative functions. As we all know, paroxysms are often induced by such emotions as anger, or joy, or fear; by the outbreak of a thunderstorm; by being left alone in the dark. They may attend agoraphobia or claustrophobia. In addition to such direct causes, acting through the central nervous system, we have to bear in mind that agencies acting directly upon the peripheral nervous system may induce attacks. Heberden has left on record an account of asthma induced by the pressure of an exotosis, pressing upon the vagus nerve; while Gairdner has likewise described attacks dependent upon the growth of a neuroma in the same nerve. All these may be regarded as examples of a direct influence on the nervous system, inducing asthma.

The reflex causes are multifarious, and act through almost every part of the body. The most important of these are concerned with the respiratory mucous membrane, whether nasal, laryngeal, or bronchial. The irritation of dust, fog, pollen, scent, and even of cold air, is universally recognized. It has long been known that various nasal affections are powerful agencies in producing asthmatic paroxysms; but the fact that they do not all do so is quite sufficient to let us see that there must be some primary neurotic condition as the basis of the affection. The other important fact—that hay fever is not always attended by asthmatic paroxyms—leads exactly to the same conclusion. Many an unfortunate sufferer has passed through a long series of nasal operations, only to find, at the end, that the asthmatic paroxysm was not absolutely dependent upon the condition of the mucous membrane.

The digestive tract — whether faucial, pharyngeal, gastric, enteric, or even rectal—is so thoroughly recognized as a powerful factor in the production of the asthmatic paroxysm that it is quite unnecessary to do more than mention the series. The urinary tract, both renal and vesical, undoubtedly acts in certain cases in a similar way. Occasionally cases have presented themselves before me in which mobility of the kidney—a subject which is no stranger to a Birmingham audience

—has led to a paroxysm. In a manner entirely analogous, pelvic disorders, both ovarian and uterine, may produce an attack. The skin, lastly, amongst the more common causes of a paroxysm, cannot be neglected, as chilling of the surface has been known to lead to severe attacks.

The counterfeits of asthma, or the varieties of paroxysmal dyspnoea not dependent upon a real neurosis, are for the most part toxic in origin. It would serve no useful purpose on this occasion to enter upon even an enumeration of these; but it may be well to mention three of the most common: the dyspnoea (1) of diabetes, apparently brought about by an acid intoxication; (2) of uraemia, undoubtedly induced by the retention of many products of metabolism; and (3) of cyanosis, certainly produced by faulty aëration of the blood. In all these forms of dyspnoea and their congeners there are clear points of distinction, not merely in the ordinary clinical features, but in the results obtained by modern methods of investigation, which serve to effect a diagnosis. It is well known, for example, that in true neurotic asthma, as a general rule, there is a considerable increase in the number of the eosinophiles—an increase which is not found in the forms of secondary dyspnoea.

To sum the matter up, asthma, in my opinion, is due intrinsically to a spasm of the smaller and smallest bronchial tubes, brought about primarily as the result of a neurosis. Such a restricted pathology may, however, be too narrow to suit every case, as the condition of spasm may spread widely throughout the respiratory apparatus. We can with our own eyes see the many efforts made by Nature to overcome it in the movements of the nose, the cheeks, the neck, and the chest. In many cases there is undoubtedly, in addition, a catarrhal condition of the respiratory tubes, as evidenced by the presence of Curschmann's spirals, and the cellular material in the expectoration. The great point, however, is that the central fact lies in the bronchial spasm, and that experiments in the physiological quadrupeds provide us with a complete picture of the asthmatic paroxysm.

With your permission, a brief reference may be made to one aspect at least amongst the many sequels which follow repeated attacks of asthma. As is universally recognized, a paroxysm leads to overdistension of the lungs; and repeated attacks induce emphysema, at first of the marginal, but later of the universal, variety, attended by sclerosis of the pulmonary blood vessels and dilatation of the heart. How is the overdistension brought about? It seems to me that the researches of Brodie and Dixon have thrown a brilliant light upon this matter. From time immemorial it has been known that the expiration during the paroxysm is unduly prolonged; and some of the tracings which are now shown bring this point out very clearly. But, along with this, the fact has perhaps not obtained sufficient recognition that the inspiration is extremely forced; and in the remarkable work of Brodie

and Dixon already mentioned it is urged that inspiration, being so much more powerful than expiration, and occurring, as it does, before the respiratory phase has come to an end, must of necessity lead to overdistension of the lungs and its consequences. There is in this a very interesting suggestion. For many years the expiratory theory of emphysema of Mendelssohn and Jenner has been very widely accepted in preference to the inspiratory theory of Laënnec and Gairdner. This is not the place to discuss the matter in full, but you will perhaps allow me to state that in my own view the investigations of Brodie and Dixon have gone far to show us that, after all, the inspiratory theory cannot be summarily dismissed, as has so often been done in modern times.

There is one last point to which you will perhaps allow a brief reference. It is commonly said that in asthma the arterial pressure is above the normal. My present house-physician, Dr. Burrow, has kindly looked up my records of the asthmatic cases which have been in my wards during the last five years, and he has found that in only one did the pressure reach even what we might call a moderate degree. This was in a man of 50, who showed a systolic pressure of 140 and a diastolic of 100 mm. Hg. In no other case did the pressure exceed 120 systolic and 90 diastolic, while in one it was actually as low as 105 systolic and 93 diastolic. During the paroxysm the arterial pressure certainly rises; but it is a matter of considerable difficulty to get accurate readings at such a time, and, indeed, we must all naturally hesitate before troubling a sufferer when in the throes of an attack. One patient (a lady of 78), who was sent to me recently by my friend Dr. Guthrie Rankin, had suffered from true neurotic asthma since her girlhood. In her case the sphygmomanometer showed a diastolic pressure of 170 and a systolic pressure of 220 mm. Hg. Although there was no renal complication, the impression was left upon my mind that the high pressure was the result of a number of factors operative in elderly people, and indeed, the large heart which the patient manifested was proof of senile conditions.

We are now brought face to face with the second part of the subject, *the treatment of asthma*. As regards the general basis of management, we may assume that it consists in carefully watching over digestion and assimilation, in maintaining circulation and excretion, in obviating nasal irritation and bronchial catarrh. The details of management in every individual case involve the careful consideration of climatic, dietetic, and medicinal regulations. We must always be in a condition of baffling uncertainty as to the type of climate which will probably be most helpful to an asthmatic patient. We know only too well that many a sufferer from this affection, whose life is absolutely miserable when he lives in a clear, dry atmosphere, upon a porous, gravelly soil, at once loses the tendency to the disease when he moves into a damp, foggy town built upon a cold clay soil. Such knowledge as this will always

give us pause when we are attempting to lay down rules for the guidance of our patients. Each case must simply be treated empirically in accordance with its own requirements.

Turning to the dietetic regulations we find ourselves on much more certain ground. There cannot be a doubt that the one great principle to follow is to prevent our patients from using much fluid along with meals. My own experience has taught me that it is well to begin the day with a tumblerful of hot water or a breakfast-cupful of weak China tea, and to let breakfast, an hour or an hour and a half afterwards, consist of food which is not diluted to any considerable extent, and which is only accompanied by a small cup of tea or coffee. In the middle of the forenoon a tumblerful of water, or any other bland fluid which the patient may prefer, is to be given. Luncheon, once more, is to consist of dry food, with not more than a claret-glassful of fluid. In the afternoon the patient is allowed to take as much weak China tea as is desired. Dinner is to conform to the same principles as luncheon; and late at night a tumblerful of fluid, hot or cold, according to the season of the year and the preference of the patient, may be given. The one great principle throughout the whole dietetic regulations is to try by every means to prevent any distension of the hollow viscera.

The drugs which have been employed both to ward off paroxysms of asthma and to relieve them when they are present form a large and imposing array. First amongst those remedies which are of use from the prophylactic point of view are all remedies which aid the digestive system. Amongst this group we have, particularly, the intestinal antiseptics, such as salol and naphthol. Good though these are, their efficacy is surpassed, in my own personal experience, by the employment of calomel, combined with acetozone, in doses of one-twelfth and one-half of a grain respectively, three times a day. With these drugs, and careful supervision of elimination, the alimentary tract can be kept in the most favourable condition. Amongst drugs which undoubtedly exercise an influence which may almost be termed specific in warding off attacks of asthma, the iodides have long justly stood in the foremost place. We understand to a considerable extent how the iodides act in high arterial pressure, and in organic angina pectoris; but the exact method of action has still to be explained in regard to asthma. It occasionally happens that the efficacy of these drugs is increased by the addition of arsenic—just as we find similar assistance when treating arterial diseases.

In the treatment of a paroxysm, our methods will always be largely guided by what we know from physiological experiments. We have been taught by the results of investigations that muscarine, for example, can produce paroxysms of asthma in the ordinary animals employed in the laboratory which present a perfect resemblance to those seen in the disease. The

forced inspiration, the long expiration, the rhonchi, the distension, the cyanosis of the mucous membranes, the cold dampness of the skin, the staring eyes—all these may be produced at will by the employment of muscarine. On the other hand, belladonna and atropine, chloroform and ether, opium and morphine, put an end to such paroxysms. These are just the drugs which we all recognize as being helpful in cutting short the paroxysms of asthma in our patients. Lobelia, in the form of the ethereal tincture, has long been regarded as almost specific in its influence over the paroxysm; and the researches of Brodie and Dixon have shown that this drug has a powerful influence in experimental asthma, although its influence is shorter in duration than that of some of the remedies which have just been mentioned. Grindelia has been used by me for more than thirty years; but, on the whole, it has seemed to me to be less powerful than lobelia. The whole series of the nitrites is of immense value. Even so weak a preparation as spirit of nitrous ether is useful. Nitro-glycerine, erythrol tetranitrate, and sodium nitrite, all are to be most warmly commended for employment during a paroxysm of the disease. Perhaps you will allow me to express one warning note as regards the employment of cocaine. In many cases, instantaneous relief is brought about by the use of various sprays which contain this remedy. The relief which they often produce is so great that a patient is insensibly led on to use more and more of the remedy until it has completely mastered the will and the patient falls a victim to the cocaine habit.

Two methods of treatment need only be referred to in this place—one is the use of different forms of powders which, when burnt, gives off dense fumes. The basis of most of these is an aromatic herb combined with an alkaline nitrate. Even in faraway cottages in the Highlands, we may see homely remedies of this kind devised by the wise women of the district; and no doubt every one of us has witnessed the benefit produced by such means. The other (and last) point to be brought before you is that, in a large number of cases, but not in all, a paroxysm may be cut short by the direct application of cocaine and adrenalin to the sensitive spot, far back upon the nasal septum; just as treatment of that region by the rhinologist during the interval is frequently followed by complete relief of the patient.

DISCUSSION.

Professor SAUNDBY (Birmingham) said he did not propose to discuss the mode of production of the asthmatic attack further than to say that all the evidence pointed to its being a reflex spasm of the inspiratory muscles, chiefly the diaphragm, and that the origin of the reflex might be anywhere in the respiratory or alimentary mucous surface, or even in the skin. The practical importance of accepting this view was that each case of asthma required

a thorough investigation of the structures concerned, in order to detect any lesion which could be remedied by treatment. The brilliant results which often followed the removal of polypi in the nose afforded a good example of the benefit that might follow treatment based upon such examinations, but in many cases of asthma all that could be said was that the respiratory mucous membrane was unusually sensitive, as there might be no definite lesion—nothing that afforded an opportunity for local treatment, and the agents that determined the irritation might be obscure, difficult to define precisely, or even unknown. Of the last a by no means uncommon example was that due to locality. Every one had met with these cases, of which Trousseau's example of two brothers, one of whom could live on one side of the harbour at Marseilles and the other on the other, might be quoted. Some years ago a little boy, suffering from asthma, had been brought to the speaker; his father was asthmatic, and on his account the family had recently changed houses, moving two or three miles to a higher situation, with benefit to the health of the father, but the little boy had developed asthma at the new house! It was often possible to get rid of asthma altogether by such a change; or, conversely, there were people who never had asthma except in certain localities, as at a certain elevation or on the sea level or actually at sea. In these cases the particular factor that determined the occurrence of the attack was difficult to understand. The emanations of animals and furs, although undoubted, were also obscure in their nature. Some years ago a young woman was brought to the speaker who had only recently suffered from asthma, and on going into her history he found that her people kept a furrier's shop in the middle of Birmingham, but lived in a suburb, and it was only since she had left school and begun to take her share in the work of the shop that she had suffered from asthma. The attacks ceased when she found another occupation. The emanations of certain flowers or of straw were equally capable of setting up an attack, but what the particular agent was was unknown. Fortunately, however, in these cases the exciting causes of the attacks were roughly known and could be avoided, but there were many others in which the most careful investigation failed to show any definite cause. It was quite usual to incriminate various articles of diet, and unquestionably it was most desirable that asthmatic patients should live a simple, life and that the last meal in the day should be small and easily digested, but, in spite of all these precautions, attacks of asthma occurred, sometimes succeeding one another so frequently as to reduce the patient to despair, and then happily they might disappear and the patient enjoy an interval of freedom as unexpected as the previous incidence. Asthma was, as a rule, developed in early life, and was distinctly a family disease, but it might be acquired, and the speaker had met with several cases which had developed in people past middle age, in consequence of influenza. The

treatment of asthma, when the cause could be discovered, was as satisfactory as it was brilliant, but in the other case it was neither the one nor the other. Careful dieting, the use of iodides and fluid extract of grindelia robusta tended to diminish the frequency and severity of the attacks. The various asthma powders should be burnt in the bedroom every night when an attack was anticipated; a spray of atropine and cocaine was valued highly by some patients and in others seemed to be of little service; adrenalin was also worth trying, and in some appeared to do good. But these remedies often owed their reputation to the alternations, to which allusion had already been made, where periods of freedom followed periods of liability to the disease, without any precise explanation being forthcoming of the change, and independently of the action of any drug.

Dr. Samuel West (London) said that while he accepted the view that asthma was a neurosis, the question was where the seat of nerve excitability existed. The asthmatic attack might, it was true, be excited by a number of different peripheral irritations, but only in a person who was already asthmatic. The analogy with epilepsy was close, and just as epilepsy depended on some central nerve irritability, so did asthma. Asthma, like ordinary epilepsy, developed in childhood, and if a patient were not asthmatic when young he was unlikely to become so in after-life. In other words, if an adult developed some form of paroxysmal dyspnoea, it was probably not true asthma, but one of the many counterfeits to which Dr. Gibson had referred. The curious periodicity to which reference had been made was similarly seen in epilepsy. After all, the first essential in the discussion of asthma was correctness of diagnosis. Plastic bronchitis, for example, frequently led to attacks of paroxysmal dyspnoea which might be wrongly diagnosed as asthma. In another class of case occurring in children, the paroxysmal dyspnoea was due to the irritation or pressure of enlarged bronchial or mediastinal glands. The importance of strictly defining the meaning of the term "asthma" was shown in many of the published statistics of asthma in relation to diseases of the nose. If a writer spoke of seeing some hundreds of cases of asthma in the course of two or three years, and upon that basis discussed the effect of rhinological treatment, it was clear he must have a very elastic view of the meaning of the term.

Dr. William Hunter (London) said he had nothing to add to the remarks of previous speakers, and emphasized the illuminating manner in which Dr. Gibson had dealt with the anatomical and physiological features of the disease. The actual nature of the disease was unknown. He treated his cases for the most part with potassium iodide, and, after referring to the value of nitrites, said that in his opinion the spasm present was more important than the congestion.

Dr. Stacey Wilson (Birmingham) drew attention to the fact that the more they knew of the potency of reflex disturbances, the less they were willing to say that such a condition as asthma must necessarily be due to some inherited tendency, important though this was in most of the ordinary cases of asthma. He had recently seen a case in which severe vasomotor spasm causing complete deadness of the hands and feet, and accompanied by symptoms of angina pectoris, was apparently due to irritation arising in the colon. If such spasm could be produced by abdominal irritation, it was probable that the asthmatic spasm might arise from reflex causes quite apart from an hereditary tendency. Discussing the use of nitrites in asthma, he said that it must not be forgotten that the spasm might be of a compensatory nature and of value to the patient. The spasm might be compensatory to changes in the mucous membrane interfering with ciliary action, and if this were so it would afford a reason why nitrites were not more universally successful in asthma.

Dr. William Ewart (London) laid stress upon the peripheral nature of the causation, and of the limitation of that peripheral field to the respiratory and alimentary tracts. Although the respiratory system was the main seat of the spasm, it was apt ultimately to become generalized. Another feature was the surprising variety in the kind of irritation. This was a key to the remarkable diversity of the remedies employed. With reference, in particular, to the various "asthma powders," he pointed out that all they had in common was that they had to be burnt. This, he thought, arose from the fact that CO_2 arising from combustion, which was now well known to be the prime mover of respiration, was the thing most likely to overcome the inspiratory immobility of true asthma.

Dr. Otto Grünbaum (London) said that in cases of asthma associated with chronic bronchitis, vaccine treatment was sometimes followed by distinct improvement. He referred to a case in which morphine and potassium iodide, except in very large doses, having failed to give relief, a vaccine of catarrhalis and pneumococcus was followed by improvement. His experience was sufficient to permit him to conclude that some cases improved under the treatment.

The President thanked Dr. Gibson for his address. While recognizing that it was better to use the term "asthma" in a strictly nosological sense, he thought that something might be said for those who used it in a symptomatic sense as indicating paroxysmal dyspnoea in whatever circumstance it arose. He wished to emphasize the practical importance of the conception that asthma was essentially a neurosis, a condition which was closely associated with the idea of nervous instability either general or local. Such instability might be inherited, and in such cases asthma depending on it generally came

on early in life, was liable to be excited by very slight and elusive provocation, and was likely to be more difficult to treat. When asthma occurred for the first time later in life, it was more likely to be due to some obvious peripheral irritation, and success in treating it then turned largely on the opportunity of dealing with the latter. He thought the influence of diet was to be explained by the influence of the different classes of food upon nervous stability. He deprecated the limitation of treatment to the symptomatic indications merely. More attention should be paid to causal indications.

Reply.

Dr. Gibson, in reply to the discussion, said he was perfectly willing to admit that Professor Saundby was probably quite correct in charging him with narrowing down the pathology of the condition within rather strict limits. The mere fact that some expectoration, containing Curschmann's spirals and Charcot-Leyden crystals, was often found at the end of a paroxysm, showed that there probably was a certain catarrhal element in many cases. He was greatly interested in the remarks of Dr. Samuel West, and granted quite freely that there was more than a mere bronchial spasm in a large proportion of cases. One instance which he had quite recently seen, in an American schoolboy, aged 13, seemed to him a proof of this. The patient referred to had some protusion of the left eyeball, and some dilatation of the left pupil, and he could not but regard this as the outcome of pressure on some of the ramifications of the sympathetic nerve, probably by an old tuberculous deposit. Dr. William Hunter's statement as regards the value of the nitrites in the spasm entirely agreed with his own experience, and one of the tracings which he had sent round illustrated the instantaneous relief afforded by the employment of these remedies. Dr. Stacey Wilson brought forward the interesting fact, in illustration of reflex influence, that affections of the colon might produce Raynaud's disease. This was certainly true, and he himself had placed on record two instances in which Raynaud's disease had been entirely relieved by the fixation of a movable kidney. Dr. Ewart's humorous remarks reminded him of the words of the old poet:

Non fumum ex fulgore, sed ex fumo dare lucem.

Some of Dr. Ewart's remarks brought very vividly before him the interesting observations of Yandell Henderson upon acapnia. The bacteriology of asthma, which had been referred to by Dr. Grünbaum, could only be studied in those cases in which there was distinct evidence of some concurrent catarrhal condition. Probably in the future, in a certain proportion of cases, bacteriological investigation and vaccine treatment would be helpful.