ASTHMA—A NEW CONCEPTION*

by

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Introduction

Of the relatively few emergency calls made upon you in practice, the call for treatment of the acute attack of asthma will be by far the most common. In fact with some patients the calls become so frequent and at such inconvenient moment that both the doctor and his patient begin to meet with ill grace.

The late Wilfred Trotter in an address on emergencies said this: "In a certain sense of the term the feeling of emergency can never be far from the mind of the active doctor. However disturbing that experience may be, and however much at times he may be inclined to envy the calm and prestige of more secure professions, if he is a person who prefers having a man's job to having one of the other kind, he will remember that his deficiency and his exposure are the price to be paid for that dignity." He continued, "When we turn to consider the emergencies in the strict sense of the term with which the doctor has to deal, we find we are still in a region where personality at least holds its own in comparison with technical equipment. For the more urgent the call for decision and action, the more important are character, the slowly matured power of judgment and a grasp of fundamental principle, and the less trustworthy are mere detailed knowledge and executive skill." Although Wilfred Trotter was discussing emergency from the surgical angle, these words have a very important bearing upon the doctor and the asthmatic patient. I am going to-day to talk about the treatment of asthma and to discuss with you the aetiological factors concerned, certain of which are new, being based upon recent clinical research and which, in my view, are of considerable importance in the treatment of this so-called disease.

Unfortunately there has grown up in the profession a scepticism about asthma. So many doctors have thought of it as either purely psychological or are concerned with it only because of its nuisance value in practice. Sanderson (1945) in a recent lecture said: "One tends to think that the world might be divided into those who have asthma, and those who do not; but the truth may well be that everyone has an asthma threshold, a certain greater or lesser inborn susceptibility to the con-

dition—much in the same way that everyone might be said to have a sea-sickness threshold. The individual in whom this constitutional tendency is high is the patient with clinical asthma; when this tendency is low, as it is in the majority, it is unlikely that the condition will ever occur, although such powerful irritants as phosgene suggest that nearly all are potential asthmatics. This attitude of mind has an important therapeutic implication—namely that we should regard the asthmatic, and teach him to regard himself, not as an abnormal person, but as an essentially normal person with a functional disability, within the limits of which he must learn to live his life.

"Even Sir Arthur Hurst, at the end of a lifetime of subjective and objective experience of asthma summed up his creed as follows: 'I am no believer in asthma cures . . . and regard most of the popular treatment of to-day like that of ten, twenty or thirty years ago as nothing more than gross suggestion; yet I know that every asthmatic derives much benefit from good advice. He can be taught a way of life and among other things how to avoid the exciting causes of his particular brand of asthma. How to control attacks which he is unable to prevent and how to be happy in spite of bad luck of having been born with the asthma diathesis.' " With such conceptions I disagree. From the purely economic view alone it is a morbid process which is of far-reaching importance and one demanding our further investigation.

Treatment of the Acute Attack

To return, however, to the treatment of the acute attack of asthma. An understanding attitude towards the patient's distress must be maintained. It only makes the patient worse to go in looking and feeling bad tempered and out of amity with him or her. The patient especially in the early attacks, is excited: therefore, one of the first measures in treatment is sedation, preferably by means of a barbiturate such as intramuscular injection of luminal grn. $1\frac{1}{2}$ –3 or smaller doses given by mouth. Seconal grn. 3 is often useful. Next the drug most commonly used, of course, is Adrenalin, and this can be given either by inhala-

* Based on a lecture given at the Metropolitan Hospital, E.8, on December 8, 1945, to demobilised medical officers.

tion in the type of Pag inhaler—there are many commercial preparations widely advertised for this purpose—or it can be given by means of an injection using a solution of 1:1,000 adrenalin hydrochloride. (It is best to use the natural product made by Parke Davis & Co. rather than a synthetic one.) One may give it either hypodermically in doses of ms. 5–10 or m. 15 can be drawn up into a syringe and, after putting the needle under the skin, strapping the syringe on the arm and injecting one minim every half-minute until the spasm relaxes. In very severe cases adrenalin m. $\frac{1}{2}$ may be injected intravenously every one or two minutes, but no more than three minims should be given. It is, however, associated with a certain risk of cardiac failure. Intramuscular ephedrine grn. $\frac{1}{2}$ –1 or adrenalin in oil (2 c.c.) can also be used or ephedrine grn. $\frac{1}{2}$ in tablet form may be given by mouth. Most patients, however, eventually get used to adrenalin and/or ephedrine and it is then necessary to resort to some other drug. Nicotinic acid 100 mgms. either by mouth or 50 mgms. given intravenously or intramuscularly is sometimes of very distinct value, but it does produce some unpleasant flushing on occasions. Pethidine may be given in doses of 25–50 mgms. injected intravenously or 100 mgms. intramuscularly, but I have known of two cases in which the dose was repeated within four hours and this caused respiratory arrest and death. Therefore, pethidine must be used carefully. It can be given also in tablet form, 25 mgms. every four hours. Pituitrin 0.5–1.0 c.c. can be injected hypodermically with relief on occasions. Capsules of amyl nitrite or ethyl iodide can be given by inhalation. Hot strong black coffee also helps sometimes. Oxygen is given sometimes by means of a B.L.B. mask, but it is no use giving it by means of an ordinary glass funnel held three or four inches away from the face. Hurst recommended in some cases a spray of carbon dioxide into the nose. Later, when the gaseous mixture of helium 80 per cent and oxygen 20 per cent becomes available again the inhalation of this through a B.L.B. mask will prove most helpful in relieving the cyanosis and distress of the acute attack. Then, of course, the inhalation of stramonium leaves—Potter's asthma cure—is a time-honoured remedy. If these measures fail in a really severe case one must give morphia grn. $\frac{1}{8}$ – $\frac{1}{4}$, with or without atropine gr. $\frac{1}{200}$ – $\frac{1}{75}$, but this should not be done too often as the asthmatic develops a habit for the drug. Cardophyllin (aminophyllin) 0.24 grm. in 10 c.c. can be given with good effect in some cases, taking five to ten minutes to give it intravenously. In one really severe case I have given an open ether anaesthetic, but it is difficult to do when the patient is severely asthmatical and

is rather frightening both to the patient and the relatives. Rarely, venesection of a pint of blood is necessary to relieve the congestion in a very severe and prolonged attack.

Theoretical Considerations

The treatment of the acute attack of asthma, however, is only the beginning of the story. Once the patient has been brought out of his asthmatic spasm then the next question is what further treatment can be given by the doctor. Up to the present time there has been no definite standard treatment although many theories have been brought forward regarding the causation of asthma. I will quote Davidson (1941): "The fundamental cause of asthma has been the object of a vast amount of research, both clinical and experimental. In addition to the peculiar susceptibility of certain individuals of neuropathic type and the undoubted hereditary tendency to which allusion has been made the association of asthma with various other diatheses has been observed by clinicians for many generations; it is, however, only in comparatively recent times that the recognition of asthma as one of a group of allied diseases has definitely been acknowledged. According to this conception asthma may be looked upon as a symptom rather than a disease, and we should speak not so much of asthma as of the asthmatic group of diseases. These so-called "allergic diseases" or "allergies" are essentially due to a condition of hypersensitivity in the individual, who may exhibit more than one of the several clinical manifestations of the group. These include, in addition to asthma, such conditions as hay-fever, epilepsy, migraine, urticaria, eczema, angio-neurotic oedema, and paroxysmal haemoglobinuria. The hypersensitivity upon which these symptoms depend is thought to be due to the entry into the body of some abnormal substance, in many cases a foreign protein, the actual attack being precipitated by some peripheral stimulus, which in an individual not thus sensitised would be entirely negative in its results. The theory of hypersensitivity as a cause of hay-fever and the "animal-asthmas" (horse, dog, cat, pigeon, etc.) was elaborated by Freeman, who referred to these conditions as "toxic idiopathies"; he regarded the various skin manifestations, as well as migraine, epilepsy, etc., as having a similar aetiological basis. The expression "allergic diseases" was introduced into the literature chiefly by the American authors (Cooke and others), the term "allergy" having been originally employed by Pirquet to indicate an increased sensitiveness to tuberculin introduced by some change (tuberculous infection) occurring in the body during post-uterine life.

"Practically all authorities are agreed that allergy, or as it may be better to call it, hypersensitiveness, is the basic factor in asthma, and a considerable amount of work has been done in the endeavour to trace the particular allergens in asthmatic individuals with a view to obtaining a rational guide to treatment, either by preventing contact with the substances in question or by inoculation of the patient with minimal doses of the responsible toxin in order to effect a specific desensitisation. The reaction of the skin to toxins in hypersensitive persons was first shown in connection with hay-fever, which has long been recognised as allied to asthma, and later the cutaneous reactions in asthmatics have been investigated by a number of workers. . . . The hay-fever reaction is associated chiefly with the pollen of the Timothy grass, although other pollens are occasionally responsible."

Thus there has grown up for the treatment of asthma the divine afflatus of allergy with its skin tests to the various groups of proteins and as a corollary, specific and non-specific desensitisation as forms of therapy—specific desensitisation being the injection of minute amounts of the apparently specific proteins in the hope that actual desensitisation will occur and non-specific desensitisation being produced either by the injection of 10 c.c. of the patient's own blood intramuscularly—so-called auto-haemotherapy—or by the injection of typhoid vaccine or peptone using a $7\frac{1}{2}$ per cent solution at 5-7 day intervals starting with 0.3 c.c. and increasing by the same amount until 1.5 c.c. has been given. Injection of Pollacene is another form of so-called specific desensitisation which is used extensively in the treatment of hay-fever and also in the treatment of asthma. And so it is that a multitude of treatments have grown up relating to the treatment of asthma. Quoting Davidson again: "Although the cutaneous reactions are obtainable in a very large proportion of patients suffering from the asthma group of diseases, the results of treatment carried out as a corollary of these tests have been on the whole very disappointing. The number of proteins for which tests have to be made is enormous, and of all these only a small proportion is available for testing purposes. It is impossible in any given case to make deductions of conclusive value without carrying out the skin tests on an exhaustive scale; the interpretation of results is by no means a simple matter in practice, and with every desire to avoid an *ex parte* attitude on this question we cannot but venture the opinion that much of the optimism which has been expressed in many quarters as to the aetiological and therapeutic value of the tests has hardly been justified. The same may be said in the main in regard to the

question of bacterial allergy, since the results of attempts at general desensitisation have in the long run been found to be as successful as those obtained by specific treatment, though occasionally, it must be admitted, the use of specific bacterial antigens has been attended with striking success."

Then there is another school of thought regarding asthma which places all the onus on psychology. I will agree that all diseases can be worsened symptomatically by mental stress, but that is all. Other workers have blamed the alimentary tract as the main aetiological factor: for instance, the mere overloading of the stomach may be sufficient to provoke an attack. Intestinal fermentation or intestinal intoxication also has been blamed. Other workers put all the blame on the ear, nose, and throat, especially upon the nasal mucous membrane, for it has been shown that stimulation of the nasal mucous membrane in the decerebrate animal will produce an attack of asthma, and therefore, it is thought by these workers that the nasal mucous membrane is hypersensitive and reacts violently to all sorts of stimulants producing asthma. And so we find this vast amount of experimental work thrown upon us, but we as doctors still have to treat our asthmatic patients. Up to the moment it has depended upon which particular way our sympathies have lain and with which particular workers we have been associated, as to how we treat our asthmatics, but generally the results of all these forms of treatments are disappointing. The asthmatic remains:

One more unfortunate,
Weary of breath,
Rashly importunate,
Gone to her death!

To my mind Davidson was very near the truth when he said that asthma was no longer a disease, but a symptom. The question is "a symptom of what?"

Experimental Investigation

During the past three years I have been following a new line of thought regarding the causation and treatment of asthma. When I was working in the Out-Patient Departments of two of the very large chest hospitals it was my lot to see many hundreds of asthmatics who had been attending these departments from anything from one month to twenty-five years, and I saw in most of them a very definite and constant deterioration over a period of time. "Repeat medicine"—some patients had been having Mist. Sod. Iod. cum Lob. or Mist. Sod. Iod. cum Stramonium for twenty-five years, together with ephedrine grn. $\frac{1}{2}$ t.d.s., and these medicine cards were the dismal records of our failure in the treatment of this condition.

Careful history taking is the most important part of any clinical examination. When a patient consults a doctor for asthma, he begins with asthma, as does the doctor. Accurate description of the very first attack and its preceding history gives a very interesting picture however.

In nearly every case of asthma there was a preceding history of bronchial catarrh or bronchitis. It may have occurred on several occasions or it may have lasted only a few days or even hours, *but it was present before the first attack of asthma.* There are without doubt a few cases in which the attacks are truly allergic, being associated with certain foods or external irritants, but these cases give that definite history and are exceptional. Careful elucidation of the histories of these first attacks of asthma in many hundreds of cases, using no leading questions, has convinced me of this fact that nearly all cases are associated with an attack of bronchitis. Another interesting point which struck me when taking these histories was, why was it that after some years—usually about ten—these asthmatic patients suddenly began to lose weight; often as not this was the time they had been sent into the hospital—query T.B.—the story being that they had had a cough with asthma for ten years and had suddenly lost weight. The sputum had perhaps even a little streaking or flecks of blood in it about this time, and the doctor had come to the conclusion that a tuberculous infection was present. I also noticed that in many cases the patient was in a cold sweat—it was not fear coming to the hospital—it was just the fact that the slightest exertion made him sweat. The inference was that these patients were toxic; and it must be the chronic toxæmia which produced the general feeling of tiredness, increasing ease of fatigue, sweating and loss of weight and in many cases loss of appetite as well so apparent in the asthmatic's history. And so I put those two facts together—bronchitis first and toxæmia later. The area of the epithelium of the respiratory tract is a very large one and obviously if there was an infection, a chronic smouldering infection, there would come a time when those toxins would produce some ill effects. In the old days when discharging wounds were common it was accepted that if the case went on long enough the patient would develop amyloid disease due to loss of protein. Therefore, it did not seem to my mind to be a very distant cry to postulate that if these cases were infective in the beginning and had a smouldering low-grade infection consequent upon this that this would in the end produce the very symptoms which I was seeing. The next link was that all these patients said that when they developed a cold it didn't stay in the nose—it always went to the chest and made the asthma

worse. In other words the cold would light up the low-grade infection, give them acute bronchitis, and then the asthma followed—surely there must be some relation here.

The next point to consider is, therefore, what is asthma? To quote Davidson again: "asthma is a paroxysmal condition . . . a spasm of the unstriated muscle of the bronchioles . . . produced by some abnormality of the nervous mechanism of respiration."

In normal respiration it is the vagus which is the controlling factor. By means of the Hering-Breuer reflex inspiration is stopped and expiration begun—unconsciously and rhythmically. Similarly the cough reflex is related to the afferent fibres from the respiratory mucous membrane and larynx which pass upwards in the vagi (S. Wright (1940)). Therefore, asthma must be related to the vagus in some way.

I studied these asthmatic patients through a bronchoscope and I have now seen more than a hundred attacks of asthma under direct vision. That bronchial spasm occurs there is no doubt: the small bronchioles definitely become smaller during the attack, but wherever the bronchus divides into smaller divisions the spasm produces not a circular narrowing but a transverse slit which is maintained during expiration, and it is this which causes the audible wheezing and expiratory distress. Not only are the bronchi and the bronchioles affected during an asthmatic attack, but also the trachea and laryngeal cartilages show some degree of spasm and the nasal airway is lessened. In other words, all parts of the respiratory tract, including the upper respiratory tract, supplied by the vagi are affected, due to a persistent overstimulation of the parasympathetic system.

Before discussing the possible causes of this prolonged and repeated parasympathetic stimulation reference must be made to certain other important features noted at bronchoscopy on these asthmatic patients. In every case the mucous membrane was swollen and covered with a very tenacious glairy whitish mucus, often so thick that it could be aspirated through the sucker only with difficulty. Scattered all over the mucous membrane were raised patches rather yellowish in appearance and often definitely ulcerated. In advanced cases of asthma the mucous membrane covering the larynx was also involved in this inflammatory process. The bronchi were often filled with tenacious yellow sputum, even in cases bronchoscoped during a quiescent phase and especially seen in children. In many cases swabs were taken from the bronchi and from the ulcerated areas and in all cases the streptococcus haemolyticus of the viridans type predominated. In old-standing cases the mucous membrane often looked almost leukoplakic in

appearance. All of these findings pointed towards the infective theory.

The mechanism of the normal cough reflex must be considered now. Suppose a small droplet or plug of mucus lodges in one of the smaller bronchi, the epithelium is stimulated and afferent impulses pass up the vagi to the cough centre in the midbrain. The efferents call for a deep inspiration and all the muscles of inspiration, both voluntary and involuntary, take part and the bronchi dilate due to sympathetic stimulation. Active expiration, again with the voluntary muscles taking part, is begun against a closed glottis. The pressure of air within the bronchial tree is rapidly raised and when the glottis is opened the air rushes out and this, together with the *contraction* of the unstriated muscle of the bronchioles and bronchi causes the plug to be shot out through the glottis.

To return to the consideration of the actual mechanism of asthma. If the whole of the mucous membrane of the trachea and bronchi is swollen afferent impulses must be passing up to the cough centre continuously, and so the desire to cough must be present at all times; and the persistent and unproductive type of cough is a characteristic of the asthmatic. Furthermore, inspiration will always tend to be diminished because of these afferent stimuli and so dyspnoea on exertion will be a constant feature of the asthmatic—which thing is always present.

But how or why do the asthmatic paroxysms occur? It was noted during these bronchoscopic investigations that if the bronchoscope was pushed down the bronchi until the diameter of the beak was equal to that of the surrounding bronchial tree, that stimulation would invariably produce, even with the patient in the third plane of general surgical anaesthesia, an attack of asthma. In other words, the trigger mechanism of the asthmatic attack is further afferent vagal stimulation. Asthma is in fact an aberration of the normal cough mechanism.

The effect of injecting a glutinous fluid such as glycerine through the bronchoscope, so placed that it did not produce an asthmatic attack, was next noted. In all cases an attack of asthma was precipitated which could be immediately relieved if suction was applied so removing the fluid.

The character of the mucus secretion covering the mucous membrane of the trachea and bronchi in these asthmatic patients has already been described (it can be well seen in the sputum pots of these patients also. A few minutes after expectoration the typical colourless sputum is so viscid that it almost sticks to the pot). The cause of the asthmatic spasm seems, from these observations, to be proved: *it is the tenacity of the secretion*

from the swollen and inflamed mucous membrane causing persistent afferent vagal overstimulation.

Anything which will cause further secretion to occur—such as infection with a common cold or an attack of bronchitis—will precipitate an asthmatic attack until or unless that sputum can be expectorated. This is in keeping with the clinical observations. Furthermore, the asthmatic patient knows that once the sputum “begins to break” the worst of the asthmatic attack will be over, especially if the inhalation of stramonium leaves or the injection of adrenalin solution allows him to take in breath deep enough to give one deep cough and so expectorate a little of the sputum.

Relationship of Experimental Data to Theories

Since it appears to be the vagus system which is the important factor in the causation of asthma the relationship of gastro-intestinal upset, certain foods or over-eating in precipitating an asthmatic attack appears simple to explain merely by postulating further stimulation of the vagi in the gastro-intestinal tract.

Regarding the psychological aspects it is a known fact that any chronic infection may in the end, in certain types of individual, so undermine the nervous constitution as to produce many so-called functional disturbances. The effect of successful treatment on many of these asthmatic cases on the psychological anxiety state, in my opinion caused by the disease, has been dramatic. A certain number of the cases had been subjected first to psychiatric therapy without helping them in any way. Months later the effect of the new treatment, about to be described, was watched with care. In many the psychological outlook of these patients has been improved so greatly that it appears certain that the psychological factor must be secondary to the toxæmia from which the patients were suffering.

In the introduction of this article Sanderson was quoted regarding the asthmatic effects of phosgene on normal persons. Phosgene is an irritant which causes the secretion of much mucus and exposure will cause inhibition of the cough reflex by voluntary action through fear of further inhalation. The effect will be the accumulation of tenacious mucus in the bronchial tree, causing excessive vagal afferent impulses to pass to the medulla and so causing an asthmatic spasm. Such a hypothesis is in keeping with these experimental findings here described.

It will be argued that the typical allergic reaction is swelling and oedema of the tissues. But such a reaction subsides leaving no trace, cf. urticaria.

That true allergic asthma exists there is no doubt, but these cases have a clear-cut history, e.g. strawberries, shellfish, anaphylactic reactions, etc., and are in a very small minority. That skin reactions are obtainable to multifarious protein and other substances is self-evident. But if the infective theory is a true explanation of asthma then there must be the ever-present absorption of bacterial endotoxins from the respiratory tract, and this may cause a bacterial endo-allergy which is however of secondary importance from an aetiological point of view and which may disappear if the infection is removed. Such a secondary bacterial endo-allergy would explain the allergic manifestations often associated with asthma and would also explain the disappointing results of desensitisation therapy.

The relationship of the upper respiratory tract infections to asthma also appears to be more easily assimilable. The respiratory epithelium begins at the nares and is continued down to the alveoli of the lungs. Therefore the term respiratory catarrh should be used and the whole tract searched for infection and treatment arranged accordingly.

The relationship of vasomotor rhinorrhoea to asthma is more difficult to correlate. My view at the moment, based on observing the effect of treatment in these cases, is that the relationship lies in the bacterial endo-allergy to which reference has already been made.

Dusts, pollens, smells and the like appear to activate an asthmatic paroxysm by stimulating the inflamed and sensitive mucous membrane of the respiratory tract so setting up vagal overstimulation.

Therapeutic Applications

The theory having been formulated and the experimental data attained, a new line of treatment was attempted. The early experimental therapy will not be detailed here; only the present routine treatment need be discussed.

A general clinical examination of the patient must be an essential prerequisite of therapy. Other respiratory diseases must be excluded with routine radiological examination of the chest. The condition of the heart and vascular tree must be assessed, the urine examined and the condition of the blood noted. A careful examination of the upper respiratory tract, with the aid of radiograms, is an essential in planning one's therapy. Especially should the antra and adenoid pad be observed. At the moment it is my impression that the nasal mucous membrane is often a mirror of the condition of the bronchial epithelium.

General Treatment

General hygienic measures such as good clothing, adequate fresh air, etc., are important and must not be neglected. Full dosages of vitamins A, D and C are necessary, often supplemented with Nicotinic acid 50-100 mgms, t.d.s. If there is any deficiency in the haemoglobin content, iron must be given. In children, a course of ultra-violet light during the winter months is a most valuable adjunct. Breathing exercises of the type recommended by the Asthma Research Council are of the utmost value.

Lessons in "deep coughing" as opposed to the usually ineffectual asthmatic cough are of paramount importance. Often in the early stage of an asthmatic attack a few good breaths using the diaphragm followed by a successful deep cough will abort the attack because it dislodges the tenacious sputum causing the asthmatic spasm. Of the drugs used, the iodides are most helpful in loosening the secretion. The following mixtures are useful:

or			
R _x Sod. Iod.	gr. 2	R _x Pot. Iod.	gr. 3
Sod. Bicarb.	gr. 10	Pot. Bicarb.	gr. 15
Ammon Chlor.	gr. 7	Ammon Carb.	gr. 3
Vin. Ipecac.	m. 5	Syr. Tolu.	m. 30
Ext. Glyc. Liq.	m. 15	Aq. Chlorof. ad.	½ oz.
Aq. Menth. pip ad.	½ oz.	Sig. ½ oz. t.d.s.	
Sig. ½ oz. t.d.s.			
or			
R _x Pot. Iod.	gr. 3	R _x Tinct. Lob. Aeth.	m. 10
Tinct. Stramon.	m. 10	Tinct. Bellad.	m. 10
Ext. Glyc. Liq.	m. 20	Sod. Brom.	gr. 10
Aq. Chlorof. ad.	½ oz.	Aq. ad.	½ oz.
Sig. ½ oz. t.d.s.		Sig. ½ oz. t.d.s.	

Sedative cough linctuses such as Linctus Diamorphine 1 dr. nocte or p.r.n., or the following:

R _x Tinct. Camph. Opiat.	m. 20
Tinct. Scillae	m. 5
Syr. Tolu. ad.	1 dr.
1 dr. p.r.n. sine/aq.	

are helpful.

The use of the other antispasmodic drugs has already been described.

Special Therapy

Thus far the recognised treatment of asthma has been described. I will discuss now the line of therapy which has been used during the past two years on one hundred and more cases of asthma and which has produced a temporary cure in over 80 per cent of these cases. The term, temporary cure, has been purposely coined since the longest period that any one case has been followed up is just over two and a half years. In every case in

which a relapse occurred further treatment was given with immediate benefit. Of those cases which responded in the first instance, the relapse rate over a period of eighteen months has been well under 20 per cent. The earlier in the disease process that treatment is instituted, the more likely is complete success. Children especially seem to respond dramatically.

The upper respiratory tract needs careful consideration and should be treated first. Antral washouts followed by the instillation of penicillin, 30,000 units in 3 c.c. to each antrum should be performed. The adenoid pad, if present, should be removed and nasal breathing encouraged especially at night, using if necessary a special dental stent.

The tonsils should be removed only if there is any evidence of gross infection or scarring. In children, the large soggy tonsil is best left providing the upper respiratory infection is cleared. (Ivor Griffiths (1937).)

Treatment of the lower respiratory tract is performed with the aid of a bronchoscope. Under general anaesthetic the aspirating bronchoscope is passed and the whole of the bronchial tree is aspirated carefully. If the secretion is very tenacious it may be washed out with a sodium bicarbonate solution or a hydrogen peroxide solution. When all secretion has been removed, the bronchial tree is sprayed with a solution of 1 : 1,000 propamidine (May & Baker) using 10-20 c.c. in all. Any excess of solution remaining after this is aspirated. If this procedure causes an asthmatic spasm or if spasm occurs within the first few weeks following treatment this does not indicate that the therapy has failed.

As an adjunct to the form of treatment the use of penicillin, especially when there is much yellowish sputum or gross upper respiratory infection, is most helpful. It may be given either by means of a fine spray from a Pag, Maw's or de Vilbiss nebuliser, but the patient must be prepared to use it very frequently indeed. Mixing the penicillin solution with 25-50 per cent of glycerine produces a better nebule. While inhaling the solution, regular deep inspiration with the mouth open followed by expiration through the nose will bring the penicillin into contact with the whole of the upper respiratory tract and the larynx and the trachea.

Penicillin can be given also by the intratracheal route, using 50,000 units in 5 c.c. It is my practice to give the patient six or ten such injections prior to bronchoscopy, filling the bronchi on alternate sides on successive days. (Incidentally it has often been noted that if the penicillin solution is run in too quickly an attack of asthma is produced, the spasm often being apparent only on the side filled. This finding appears to substantiate the theory already discussed earlier.) It is my experience that propamidine, however, is much more lasting in its action as an antiseptic agent, but that penicillin is a most important adjunct to successful therapy.

It is unfortunate that the treatment outlined is of such a specialised character, but the results have more than repaid the labour. The practitioner will be able in the future to take a more active part in the treatment once penicillin is more readily available, but in my opinion the bronchial suction and bronchial lavage is of the greatest importance.

Conclusion

1. The treatment of the acute attack of asthma has been described in detail.
2. A survey of the accepted theories of the causation of asthma has been given.
3. A description of the bronchoscopic findings in a large number of cases of asthma has been given.
4. The mechanism of the asthmatic attack has been discussed.
5. The relationship of this mechanism to the present theories has been apparently correlated.
6. Asthma has been shown to be a symptom of bronchitis and not a disease *per se*.
7. A new therapeutic approach has been detailed, the results of which have been successful in producing temporary cures for variable periods up to two and a half years.

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