"A Study of Asthma"

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A great deal of matter has been written during the last thirty years, with regard to asthma, which it may be advantageous to collect together and sift out, endeavour to arrive at some definite conclusions in place of the unwieldy mass of contradictory evidence which has been placed before us. It is first necessary to state clearly what we mean by the term "asthma," for up to the present century it was used to denote any kind of dyspnoea, arising from whatever cause, merely meant "difficulty of breathing." (Dyspnoea, panting.) The dyspnoea of heart disease was termed "cardiac asthma," that arising from gut or "stomach asthma," so also there were "renal," "gastro-intestinal," "osteo-muscular," "arthritis," "tuberculous," "nasal," "uterine," "thymic," "hysterical" asthma spoken of. The earlier pathologists referred the causation of the so-called asthma to any organ from affected by them on physical examination, or after death. Begin refer it to the lungs, Broussais to the heart, Heberden to the brain and spinal cord; until, soon after percussion and auscultation came into use, the very existence of the disease as an independent affection was denied by Boston orthopaedic physicians, it came to be regarded merely as a symptom of various affections. With improved methods of physical

Diagnosis however, it was found that there was a certain
class of case which could not be satisfactorily accounted for
in this manner, no organic lesion being discoverable.
This class was therefore separated from the rest under the
name of "nervous asthma"; it gradually came to be clearly
distinguished from the many forms of dyspnoea with which
it had been confused, and was found to possess features
and definite features of its own.
It is this latter form of "nervous asthma", which exhibits
itself in its pure form, merely by the occurrence of paroxysms
of dyspnoea, that it is proper to discuss in the present
paper. We shall first proceed to consider the clinical history
of the disease, under the headings of the premonitory symptoms,
the phenomena of the paroxysms, the intervals, the complications,
and sequelae.

**Clinical History**

**Premonitory Symptoms.** In many cases the approach of
the paroxysms of dyspnoea, which constitute the "asthmatic
attacks", is heralded by premonitory symptoms, which are of
great variety. Among the most common is a feeling of
great nervousness, sharpness, without anything to account
for it. This is generally felt on the evening preceding the
attack, and may be so irresistible as to cause the patient
to fall asleep over his dinners. Boyce, who wrote in the
sixteenth century, particularly notices this symptom in his
own case. "Thus appears," he says, "a great fulness of breath
and stertor."

Jolness of the head, with a slight headache, severe sleeplessness, in the evening before the fit. A peculiar itching is also very common, in some cases fleeting about all over the head and neck, but generally confined persistently to the region under the chin. Dr. Berkert describes as occurring "not infrequently, a few isolated dark blue papules, probably erythema papulatum, appearing at or near the chin, on one side of the neck, causing a great deal of itching." In one case a mother informed me she always knew in the case of her child, when an attack was coming, before any asthmatic symptoms appeared, by his persistently rubbing his eyes, "because they were itchy." There were no signs whatever of conjunctivitis or any other inflammation. Dr. Hyge Salter mentions a case in which an attack of ophthalmia always ushered in the asthma. A profuse flow of pale urine is a frequent premonitory sign, this often occurs during the early stages of the attack. I have known it accompanied by several loose motions. In some cases an unusual brightness, a peculiar excitability, dyspnoea of spirits, are always forewarning of an approaching attack. Some writers mention fluorescence tenerness, but these are probably more often the actual exciting cause of the attack. So also, cough fits of sneezing are stated to be frequent premonitory signs, but these are obviously signs


Signs of inflammation of the upper respiratory passages, which may reflexly cause the paroxysm. In many patients a cold in the head or a slight bronchitis, is quite sufficient to induce wheezing and shortness of breath, sometimes an actual paroxysm. Neuralgic pains in various regions have frequently been observed as premonitory symptoms. Deep-seated aching in the limbs, joints, frequently in the testicles, have also been noted. Dr. Hyge Salter mentions a very curious case in which the testicle, the titica, from the knee to the ankle were always affected on the same side, sometimes the right titica, sometimes the left. The same observer also describes a case of a patient who "experienced a tingling sensation from the spine, with other symptoms of nervous disturbance, extreme irritability &c., for two or three days before the attack." Thus preliminary paresthesiae and nervous disturbance of various kinds have been frequently observed; they display an infinite variety, have been likened by Dr. Hyge Salter, Romberg, others to the aura preceding an epileptic fit, these termed "asthmatic auras." They appear to suggest a condition of nervous instability, which reaches its climax in the asthmatic paroxysm, for they generally appear in the afternoon or evening preceding the attack, though sometimes one or two days before, disappear after the fit, and are not again experienced until just before another fit comes on. In many cases they

they are absent altogether, the paroxysms of dyspnoea come on quite suddenly without any warning.

The Phenomena of the Paroxysm.
Many graphic descriptions of the asthmatic paroxysm have been written, indeed no physician can long in practice without having an opportunity of observing one for himself. Once seen, it is never to be forgotten. The patient may have had his usual premonitory symptoms, or have experienced a feeling of tightness of oppression about the chest during the evening, or he may have gone to his chamber, perfectly well. He sleeps quietly for perhaps two or three hours, or longer, until, generally between two and six in the morning, he becomes restless, his breathing becomes wheezy, & finally he awakes to find himself breathing noisily and with difficulty.

The dyspnoea may now so rapidly increase that he is obliged to sit up, or stand up, at once, sleep is no longer possible; or he may perhaps be able, after raising himself on his pillows, or sitting up for a few minutes, to lie down again, to rise off for a little, only to be soon reawakened by the increasing dyspnoea, until he is wide awake & obliged to sit up, gasping for breath. Leaning forwards he rests himself on his hands, with his shoulders raised up to his ears, thus he breathes most forcibly, to give his accessory muscles of respiration every advantage. Soon however, even this is insufficient to
to
give any relief; the urgent dyspnoea compels him to
got out of bed, he starts up leaning with his hands on
the side of the bed or on the back of a chair or the edge
g of a table; or he hangs on to the mantel-piece or the
bed-post; anything to give his accessory respiratory
muscles a greater advantage. Sometimes the distress is
so extreme that he rushes to the window tears down the
curtains in his frantic struggles to get more air; and
every laboured breath is drawn with a moan, or a short-
gasping cry. His appearance is now most alarming and
horrible to behold: his face, at first pallid, soon becomes
flushed, then cyanosed, his expression is intensely anxious,
his eyes are starting and sunken; he is drenched in a cold
sweat; and each short gasping inspiration he throws his
head up, raises his shoulders still higher, while his mouth
opens and his nostrils dilate. His extraordinary muscles
of inspiration are all seen to be acting vigorously, the sterno-
mastoids especially standing out like cords. His whole being
is quin up to the fearful struggle for breath, he cannot
spake nor turn his head, takes no notice of anything.
Nor does he cough, makes no attempt to expectorate, though
he may occasionally feebly endeavour to clear his throat.
Slight somnous trididian rhonchi are audible all over the
room, the trididian far outnumbering the somnous. No
sudden sounds are all are to be heard. This extreme
Torture lasts for a variable period, generally not more
than two or three hours; it may last for fifteen to
to thirty minutes, or it maybe for several hours, or very much longer. Dr. Hyde Saltz states that he has known a patient to stay grasping the back of a chair for two days plus two nights unable to move. At this begins to subside a little, the patient gives an occasional full breath cough. This breathing now gradually becomes easier this cough strong, till he is able to expectorate a few little greyish white pellets, very viscid & tenacious. The attack is now practically at an end, relief comes rapidly & the patient is soon able to lie down again & fall asleep. He rests undisturbed till morning, when he awakes with his breathing quit easy & quiet, with no ill effects from his attack except perhaps, a little headache & feeling of weariness.

Such is the account of an asthmatic paroxysm, as seen in its pure, uncomplicated, most typical form.

**Physical Examination.**

In inspection the chest is seen to be raised & distended in the position of full inspiration, the ribs being raised to their utmost extent. During inspiration all the accessory inspiratory muscles stand into prominence, the sternomastoids & scaleni especially standing out like cords. Yet there is very little increase in the expansion of the chest. Deep hollows are seen in the supra-sternal & supra-clavicular regions, & the soft parts between the lower ribs fall in at each inspiration. This is due to the

the obstruction to the entrance of air being so great that the lungs cannot expand sufficiently even to fill up the space caused by the slight additional expansion of the chest wall. For the same reason, in severe cases, the diaphragm is drawn up on inspiration, presents with expiration, "an inverse type of action" thus being brought about.

During expiration the abdominal muscles can be seen as they contract over the chest cavity. In the least, if the hand be placed before the mouth, hardly any air can be felt to be passing. Inspiration is as a rule short and gasping, but it varies greatly, being sometimes much more labored than in others, in every case, however, it is much shorter and less difficult than expiration. Expiration on the other hand is extremely labored and prolonged, being in severe cases four or five times as long as inspiration; in the less severe attacks, the prolongation of expiration is not so marked.

Both inspiration and expiration are accompanied by numerous loud respiratory rales, but those accompanying expiration are much louder and more numerous than those accompanying inspiration. The frequency of respiration is generally diminished, being sometimes only nine or ten per minute, as a rule, it is between twelve and twenty per minute. It varies greatly in different cases, the frequency sometimes increasing. It has reached a frequency of thirty per minute. Vocal murmurs are either absent or greatly

The percussion note is hypo-resonant all over the chest; the pulmonary resonances extend beyond the normal. The chest wall thickness is diminished in extent. The auscultation the breath sounds are faint, in some instances completely masked by loud rhonchi. By far the greater number of these are resonant, showing that it is the smaller tubes that are obstructed. These rhonchi are heard all over the chest, but appear to shift about from place to place; for if one listen at a certain spot, at one time no sound may be heard, but on listening again after an interval they may in turn be numerous and noisy, as if the obstruction existed in certain tubes at one time and not at another. As a rule there are no moist rales to be heard, if any are present they are very few, in an infinite minority to the breath sounds.

The pulse is always very small, feeble, rapid, its frequency varying from ninety to one hundred and twenty. The heart can be felt to be thudding violently, there is generally some epigastric pulsation, the right heart sounds are louder than the left. The left border of the heart is within the nipple line. These facts all point to some obstruction to the pulmonary circulation.

The temperature is found to be normal. During the early stages of an attack there is frequently a profuse flow of pale urine. Sometimes they are at the same time several loose motions. The urine immediately after an attack, has been observed by Ringer to exhibit...
A considerable fall in area of oxygen chloride. In some patients the free water in the urine continues for twelve hours after the cessation of the attack, in others the urine becomes scanty, thick and cloudy, thickened with water, at the close of the paroxysm.

The Duration of the Paroxysms.

The duration of the paroxysm is very variable; it may last only ten or thirty minutes, but generally continues for two or three hours, though it may do so for much longer. In many cases the symptoms begin early in the evening, gradually increase till they reach their climax in the early hours of the morning, they then gradually pass off and are completely gone by light or noon in the morning. As a rule, in those cases in which the paroxysm comes on suddenly, the dyspnea is milder, intense spasms pass off rapidly and completely; whereas in those in which the attack is slow and gradual, the dyspnea is not so severe, but it passes off much more slowly, not so completely, a certain amount of shortness of breath persisting for some time. Frequently the paroxysms return at the same hour for several successive nights—there being complete absence of all symptoms during the day time—till they are then absent altogether for a varying period of days, weeks, or even months or years. In other cases the dyspnea continues for several days—nights with but partial remissions during the day, always becoming worse towards night, reaching its

its climax in the early hours of the morning. Some patients are never entirely free, but suffer from constant breathlessness waking up with nocturnal exacerbations, so that they are unable to lie down at night, but are obliged to sleep, night after night, propped up in a chair, perhaps for years. Dr. Faggé mentions a patient who has been obliged to do this for over twenty-five years. Some one who had not lain down for twenty years. These chronic cases however are the subjects of bronchitis, emphysema, generally also of secondary cardiac changes, which have supervened as complications of the asthma. In pure asthma there are always occasional paroxysms of dyspnoea with intervals of complete freedom, and these the general health does not appear to be in the least impaired—until after some considerable time—prolong the paroxysms are only occasional, the recovery in the intervals complete. The day following an attack there may perhaps be a little headache, but the appetite is not in the least interfered with, and all other respects health appears to be perfect, there being no signs whatever of any pulmonary disease. In all cases the dyspnoea is the least severe during the early part of the day, between eight a.m. and 11 a.m., it gets worse towards night, is most severe in the early morning. Termination of the paroxysm.

There appears to be no case on record of a fatal termination to a paroxysm of pure uncomplicated asthma.

Dr. Perini quoted five fatal cases, but all of these were complicated by severe cardiac, renal, pulmonary disease, & it is evident from his account of the cases, that death was due to these complications. It certainly cannot reasonably be ascribed to the asthmatic paroxysms.

Dr. Taylor however mentions a case of "a strong healthy young man, suffering from a severe attack of asthma, whose respirations became more and more shallow till last cease altogether; he fell forward in a state of insensibility and remain unconscious for several minutes. Artificial respiration was resorted to at once with success. By artificial respiration had been less prompt there is little doubt that he would have died." We are not therefore justified in saying that asthma is never fatal, as it is evident from the above case that it might prove so from slow exhaustion & cardiac failure in gradual suffocation. There is no doubt however that a fatal result is excessively rare.

Let us now consider what suggestions offers themselves to us from our study so far of the clinical history of asthma. We only know as yet that the disease is characterized by paroxysms of dyspnea, which may come on quite suddenly without apparent cause. That these are no signs of any pulmonary disease to account for them, that they leave no consequences behind.

1 "On Bronchial Asthma." 2nd Ed. p. 163-165.
behind.

That these paroxysms in many cases are preceded by certain symptoms, which are not pulmonary, but appear to refer to the nervous system, for the whole suggest a condition of nervous instability. By an physical examination we know that the prominent cause of the dyspnoea during the attack, is some obstruction to the passage of the air in the smaller bronchioc tubes, that this obstruction impairs expiration much more than inspiration. We know also, by the tenuity of the sounds, labored expiration, except in quite inalinating amount—that it is a dry obstruction not a fluid one, that it does not appear to be conjunct to any set of tubes, but affects the same tubes at different times in all parts of the lungs. Yet, through there must be a large amount of obstruction to cause such severe dyspnoea, this dyspnoea subsides rapidly and completely within them having been any removal of the obstruction in the mean time by expectoration, for the small amount of sputum which has been got rid of could only have blocked a few tubes results certainly not accounts for the severe dyspnoea.

We know also, by the cyanosis of the small feet, pulse, though the heat is acting violently, by the exaggerated right heart sounds by the epigastric pulsation, that there is some obstruction to the pulmonary circulation. This is all the higher so far we can throw on the subject.

We are enabled however to come to one definite conclusion.
Conclusion, that is that the dyspnea cannot be due to any excitation blocking the lumen of the tubes, for this dyspnea often comes on quite suddenly, before any excitation could possibly take place to sufficient extent to cause obstruction, subsides completely rapidly without any removal of excitation, to speak of. Moreover the dyspnea has always subsided to some extent before any cough or expectoration takes place. The dryness of the bronchi, also, the peculiar character of the respiration, the other physical signs are inconsistent with the presence of an excitation in any quantity in the bronchial tubes.

For these and the above stated reasons we conclude that the obstruction is not due to any bronchial excitation.

Clinical Types.

Clinical we meet with asthma in several different forms.

First is asthma uncomplicated by any other disorder, characterized solely by the occasional occurrence of paroxysms of dyspnea. This form is met with in its most typical character in children, before complications have had time to supervene. The attacks frequently come on in the early mornings, or maybe induced by a slight chill, damp weather, or indiscretion in diet. Secondly there is a very common form which is associated with bronchitis, conveniently termed "bronchitic asthma." In this the patients are generally older, whenever they have an attack of bronchitis, suffer from asthmatic paroxysms as well.
there may be mere or less wheezing dyspnoea constantly present, without any actual severe paroxysms. These patients suffer more in winter than in summer, as they are then more liable to attacks of bronchitis. Another distinct type is termed "hay-asthma" or "hay-fever." This is a peculiar and severe paroxysmal coughing with violent fits of sneezing, associated with paroxysms of asthma. It was called "hay-fever" because the pollen of certain grasses was the first cause of the affection to be observed. The peculiar coughing often exists independently, without any associated asthma; there are many other causes besides hay, "paroxysmal coughing" would therefore be a more accurate term. So also for that form which is associated with asthma, "coughed asthma" would be a simple and accurate designation, the term "hay-asthma" being misleading and incorrect as implying only one cause. The relation between asthma and "hay-fever" will be more fully considered later.

Lastly there is the form of asthma which is met with in the standing cases, which we may call "chronic asthma." The subject of this suffers from constant wheezing and shortness of breath, with frequent exacerbations. There are nearly always also some degree of pulmonary disturbances, which have been induced by the oft-repeated paroxysms, viz. chronic bronchitis, emphysema, and dilatation. Hyper trophy of the right heart, with, in the worst cases, general venous congestion.
Pathological Anatomy.

Regarding the morbid anatomy of asthma, uncomplicated by other disease, nothing definite has been obtained. The results of all the autopsies that have been made published up to the last thirty years have been collected by them, but these merely show that paroxysms of dyspnea may be associated with very many different diseases. Since that time only six post-mortem examinations have been described. The first by Virchow, of "an old man of 76, who had suffered from chronic bronchitis with asthma, in whom embolism of the pulmonary artery was found." The second by Ebhler, of "a man aged 59, who had for more than 30 years been subject to hay fever with chest symptoms, died suddenly on leaving a very hot sulphur bath. The lungs were found distended, the trachea bronchi filled with a frothy fluid, the mucus membrane red and swollen, the heart hypertrophied and covered with fat."

4. "Der typische Fiebersommer - Katarh. 1862. p. 69 et seq."
The third by Bambergel, of a strong healthy labourer, age 36, who presented symptoms of tetanus, — loss of consciousness, distortion of the face, opisthotonus, and tonic contractions of the muscles of the limbs, — symptoms which were warded off by a kind of epileptic cry, twice accompanied by intense dyspnœa. He died in one of these attacks; the lungs were found congested, the heart hypertrophied, and dilated. The fourth is by Trautgol. 2 of a young man, asthmatic for many years, in whom was found extensive bullous emphysema, a portion of the upper lobe of one side having developed into a large sac containing air. The fifth is described by E. Heythun. 3 The case of a baby aged 40, who had from earliest infancy been subject to typical attacks of asthma. Her constitution had been undermined by carelessness. She was also, in the highest degree addicted to the use of morphia. The disease in her case assumed of late an unusual intensity, was complicated by dyspnoeic concomitants, which indicated the near approach of the end. The lungs were emphysematous. The respiration was jumbled, ronchali ronchi were heard all over the chest. She had daily several paroxysms of dyspnœa, which presented the characteristic signs, only replaced by muscular effort. The sputum was very rich of a grayish-white colour, copious, and contained, almost always


always

long whitish fibrous threads, but very few granular tubular masses. Death took place during a prolonger attack with
cyanosis & constic. The autopsy performed 6 hours after death,
showed extensive vesicular emphysema. Both lungs were very
large, overinflated, pale, other margins were partially
covered with large bullae. The bronchi were not dilated, their
mucous membrane was red, but not materially altered;
but layers of a coarsely granular, apparently amorphous
substance, which considerably restricted their calibres, firmly
adhered to the surfaces of their mucous membranes. In one
bronchus the excitation was annular, with only a narrow
slit in the centre, in another it had somewhat the shape
of a screw, leaving a semilunar passage."
The sixth case is described by Dr. Berkan. It is quite
inconclusive on account of the severe pulmonary cardiac
complications, which are obviously the cause of much of
the condition described. Yet on this single case Dr. Berkan
forms his whole theory of the pathological anatomy pathology
of asthma. In the case in question there was a history of
phthisis, the patient had always been subject to "colds," and
in the last 14 years she had been subject to severe
attacks of bronchitis, which confined her to her for several
weeks at a time, recovery being always imperfect. Seven
months before her death, her legs began to swell, her urine
was diminished and contained albumen, she was laid up in
hospital for 4 months. "An area of the legs affected with
in Bronchial Asthma," 2nd Ed. p. 73 et seq.
Agyanosis of the face, persisted however, two days before her death, all the symptoms got worse. She passed less water, the anasarca + agyranosis increased, there was no pulse to be felt at the wrists. At the post-mortem examination there were joint hypertrophy + dilatation of the heart, irregular dilatation of the smaller bronchi, emphysema, diffuse suppuration of the epithelium of the bronchi + fibrous exudation within some of the bronchítides + alveoli, partial or complete obstruction of both small large tubes by masses of debriites composed largely of streptococci. There were also ascites + amylolysis degeneration of the liver, spleen, kidneys.

This case is more fully discussed under the head of the pathology of the disease + enough has been said for our present purpose, it is evident that all these six cases are quite inconclusive, if a point is made concerning whatever regarding the pathology of asthmaic paroxysms. This question can only be studied in pure uncomplicated cases. In long-standing cases there is found a marked hypertrophy + thickening of the muscular fibres in the walls of the smaller bronchi; the air-vesicles are joint dilated, there is hypertrophy of the muscular fibres round the interlobular openings; all the evidences of emphysema are present. There is generally also some hypertrophy + dilatation of the right side of the heart. The only conclusion we can arrive at is that the astmatic paroxysms are associated with a peculiar fibrous exudation into the smaller tubes, but that this exudation is the factor...
The Relation of Asthma to Hay-fever (Paroxysmal Coryza)

The connection between asthma and paroxysmal coryza is very interesting and instructive. Certain people are liable every summer, to a peculiar very troublesome catarrh of the conjunctiva, nasal passages, throat, with profuse watery discharge from the eyes and nose, paroxysms of sneezing. The submucous tissue of the nasal passages becomes so congested and swollen, that then human is soon almost completely obstructed that any air can pass through. This peculiar catarrh in some patients associated with paroxysms of asthma. By far the commonest cause of this affection is the pollen of certain grasses, especially hay-grass, of which the air is full in early summer in the country. This was what first drew attention to the condition, hence the name "hay-asthma.

In further study of the disease, however, it was found that there were numerous other excipients which could produce it, quite irrespective of summer hay. Moreover, that the excitant, whatever it happened to be, in one person...
person would produce "paroxysmal coryza," in another "coryzal asthma," that is, paroxysmal coryza associated with paroxysms of asthma, in a third, asthma alone. It was further observed that the excitants in the vast majority of persons were quite incapable of causing coryza, or asthma, or any irritation whatsoever. Hence it was apparent that there must be some peculiar predisposition in these individuals who suffer from them, as the exciting causes were, of themselves quite inadequate to produce any irritation. The essential cause of the coryza or the asthma then appears to be an irrisynepatic hyperaesthesia of the respiratory passages, a functional neurism which renders the respiratory tract in these predisposed persons abnormally sensitive to certain sources of irritation, which to ordinary people were imperceptible. In persons with this peculiar irrisynepasy there is no limit to the number, caprice, or utter unwarrantableness of the exciting causes, which in one case will produce paroxysmal coryza, in another coryzal asthma, simple asthma in a third. It is thus evident that this hyperaesthesia in some cases concerns the upper respiratory passages only, in others is limited to the lower ones, in others implicates the whole respiratory tract; the paroxysmal coryza merely being the expression in the nasal passages, of the same irritability which in the bronchial tubes expresses itself in some way or other in paroxysmal dyspnoea. That is, that both paroxysmal coryza and asthma are due to a functional neurism, an irrisynepatic hyperaesthesia,
hypoaesthesia, of some part of the respiratory nervous system, which gives expression to itself in paroxysms which may be excited by various thrilling causes. There are many facts which point to this intimate connection between the two diseases. Paroxysmal corryza is commonly hereditary; Sajoné states that in 35% of all his cases there was well-marked evidence of heredity, — the same is also the case with asthma; moreover, the same observer also notes that 42% of his "hay-fever" patients had asthmatic relatives. Dr. Graville Macdonald observed the same connection. Describes the case of a mother and two daughters who suffered from "hay-fever," while a third daughter suffered from paroxysmal sneezing all the year round, and a son was the subject of asthma. I know of a similar case, in which a mother was subject to asthma but had never had paroxysmal corryza; her eldest son suffered from paroxysmal sneezing, the second from paroxysmal corryza, the third from asthma. It has been pointed out by Dr. Beart of New York that nearly all sufferers from paroxysmal corryza are of a neurasthenic temperament, a fact which has been further confirmed by Dr. Graville Macdonald, — such is also prominently the case with asthma, as will be pointed out.

Both affections are much more common in males than in females. In both the tendency may be considerably lessened.

2. loc. cit.
by constitutional treatment, which raises the tone of the nervous system increases its resisting power. The utter inadequacy of the exciting causes is a further point of resemblance, and suggests their nervous character.

Dr. Macdonald narrates the case of a lady whose attacks of hay-fever were brought on by wearing except horses, who always went out to dinner in great trepidation for fear they should be horses on the table, in which case she would have to leave the room before dinner was over. Sir Hull MacLachlan also relates a case of a lady who was so sensitive that even the sight of an artificial rose was sufficient to excite an attack. Certain animal emanations will in some persons excite an attack of paroxysmal conyza, in others an attack of paroxysmal dyspnoea. Dr. Macdonald mentions cases in which the mere entry of a cat into the room was sufficient to induce a violent attack of paroxysmal conyza, relates several stories of gentlemen who found it impossible to drive a horse, on account of the same attack, the odor of the animals brought on. In other persons, equally remarkably, these emanations cause attacks of paroxysmal dyspnoea. Similarly also intra-nasal disease, whether in the form of polyps, hypertrophy of the inferior turbinate bones, retraction of the septum, or adenoids, is a very frequent cause of hay-fever in some persons, so of asthma in others.

Sir Andrew Clark considered that besides a mucous diathesis there was a local peculiariry of the nasal mucous membrane which lead or nearly to swell through vascularity. Heel, Hawthorn Hall considers that in all cases of hay asthma there is a main condition of the nasal mucous membrane, namely, chronic hypertrophic rhinitis. But Dr. Macdonald, Sir Archibald Mackenzie and Brown have shown that this is by no means the case, but that the pathological condition of hypertrophy of the septum and inferior turbinate bones has been the consequence of the reflex inflammation that the cause, the starting point having been merely the sarco-mucous hypertrophic, no apparent malitia condition being present. Hence "hay fever" and asthma are merely the expression of a functional nervous, which in the upper respiratory passages gives expression to itself in paroxysmal coughing, in the bronchial tubes in coughing some catarrh which causes paroxysmal dyspnoea. A suggestion here at once occurs to us; may not the nervous be of a sarco-motor nature, may not the dyspnoea be due to the occlusion of the smaller tubes by a vascular swelling, hypertrophy of the bronchial mucous membrane, exactly similar to that which takes place in the nasal mucous membrane? If this were so however, there should also be free evolution into the tubes, one should have numerous mucous riles, and cough and expectoration of a quantity of fluid. It maybe
be said that this hyperemic swelling might exist for some time before any sensation took place, as in the first, congestive, stage of bronchitis before any secretion takes place. But we know that in a bronchitis there is very considerable hyperemia or swelling of the mucous membrane, but yet this is quite inadequate to produce the severe dyspnea of asthma, what dyspnea it does produce is of a different character. Moreover this hyperemic swelling could not come on so suddenly or subside so rapidly, as is the case with many attacks of asthma, for instance those sharp, short, sudden attacks of purulent dyspnea induced sometimes by sulfanilamine, or ipecacuanha. Hence for these reasons we must reject this vasomotor theory of hyperemic swelling as being unsatisfactory. Already many facts strongly suggest that asthma is essentially nervous in its nature—the condition of nervous instability which is indicated by the premonitory symptoms; the utter ineffectiveness, or seen apparent absence, of the exciting cause in many cases; the absolute suddenness of the attack which is often seen, and its sudden cessation; lastly the intimate connection between asthma and parasymptomatic cyanosis—which appears to be almost certainly neurotic—all point to a nervous cause. We shall see afterwards that there are many other facts which confirm us in this view. The symptoms are inconsistent with the theory of vascular dilatation and hyperemia of the bronchial mucous membrane being the cause of the paroxysms of dyspnea, we must seek some other
other explanation. A very plausible one at once presents itself. We know that these muscular fibres circularly disposed round the walls of the bronchi, that these, in the smaller tubes, bear a considerable proportion to the size of the tubes. We know also that in these small tubes there is no cartilage in their walls, that therefore, if these muscular fibres were to contract firmly, they could very considerably diminish the lumen of the tubes. Hence may not the asthmatic paroxysm be due to spasm of the contraction of these fibres? This would satisfactorily explain the severe breathlessness, the absence of cough, of expectoration, till the close of the attack, when we should expect that the congestion instest would have produced some expectoration. It would account for the rigors of the sounds, their chill-like character, for their apparent fluttering from some tubes to others. If the spasm might relax in some tappens or others, it would account for the cessation of dyspnea without the removal of any obstruction by expectoration; in frequency there is practically no expectoration, certainly not sufficient to cause obstruction to more than a few tubes; for the utter absence of any signs or symptoms in the intervals between the attacks.

We must now proceed to consider the various phenomena more in detail, for many facts have been mentioned which require explanation.
Phenomena Considered in Detail.

Time of the Attack. The remarkable frequency with which the paroxysms occur in the early hours of the morning, towards explanation. Many patients never have an attack at any other time, in all cases the hypnosea becomes more severe, at this period. Dr. Hyle Salter explains this fact in the following manner: 'The reasons,' he states, "are two, first, the horizontal position of the body, and secondly, the greater facility with which sources of irritation meet any causes of reflex action, operate during sleep than during wakefulness, i.e., the greater propensities to reflex motor action." 'When a person lies down to sleep, the recumbent position favours the efficiency of blood to the right side of the heart, therefore to the lungs, in addition to this, the position of the body places the muscles of respiration at a disadvantage, especially the diaphragm, against the inner surface of which the recumbent position brings the contents of the abdomen to bear; it thus may alter the immediate rate at which the vital changes go on during sleep, slanting the lower sensibility of sleep, which prevents the arteries into which the respiration maybe getting from being at once appreciated. The position of the body, the tossing of sleep, soon throw the lungs into arteries they become congested; this goes on for some time gradually increasing without producing any particular effect.

but by and by this pulmonary congestion reaches such a pitch that it becomes itself a source of great local irritation and gives rise to asthmatic spasm. Now there is no doubt that recumbency is an unfavorable position for respiration; it tends to cause a certain amount of pulmonary congestion, which would be quite sufficient to initiate a paroxysm, if this were imminent, just awaiting any little opportunity. For many asthmatics can only sleep in a sitting posture, others again always require an extra pillow or two to raise their shoulders, as if they go to sleep with a low pillow they are sure to have an attack. Others again can sleep on a sofa, though not so well; this is probably because the heart is higher than feet, the right sofa affords a better support for the arms, so giving greater power to the extraordinary muscles of respiration, so that they are able to overcome any little tendency to congestion almost without effort. Certainly all asthmatics sleep better in a more or less upright position. Very often in those cases in which the patient wakes up in the early morning with wheezing and a little shortness of breath, if he sits up for five or ten minutes, the symptoms soon pass off, he is able to lie down again and sleep comfortably perhaps till morning, whereas if he persists in lying down he continues wheezing till the morning. Dr. Salter's second reason with himself, namely, "the greater facility, which sources of irritation incident any causes of reflex action, operate
"Turning sleep," — is erroneous; it is refuted by physiological authorities. "There is diminished excitability of the whole nervous system during sleep, whereas excitability is diminished, much more powerful stimuli are necessary to produce any given effect," than during wakefulness. In support of his theory Dr. Hysle Salter quotes the following case.

"We know," he says — "that the greater prominence of excitatory action during sleep has to do with it, because some asthmatics do not dare to go to sleep after the commission of any injurious, whereas they may quit its of any irregularity with impunity, if they only keep awake for some time afterwards. I know one asthmatic who often sits up late the night after taking a supper, (breathing perfectly freely) because he knows that if he goes to sleep his asthma will come on immediately, but by thus keeping awake till his supper is fairly digested, his stomach emptied, the source of irritation thus removed, he may go to sleep fearlessly, have a good night's rest." Surely Dr. Hysle Salter's inference from this case is erroneous. The obvious explanation is that if a person go to sleep too soon after a supper, the meal lies undigested in his stomach for a long period and quite sufficient to reflectively excite an asthmatic attack. For during sleep the movements of the stomach cease, digestion is retarded (Busch.) But if he keep awake till the meal is digested then he may go to sleep fearlessly.

is, the exciting cause of the attack is removed. The recumbent position, however, does not account for all cases, for though in most patients the attacks come on after they have been some time in bed, in others they occur in the evening, in others just after getting into bed, in others on waking in the morning. There is apparently in each case a tendency for the attacks to occur always at their usual period. The length of the interval between the attacks varies greatly in different cases, but tends to remain constant in the same case; that is, there is a tendency to periodicity in the recurrence of the attacks. Many patients suffer every night, more or less; others have an attack about once a week; others only at long intervals, once in 3 or 4 weeks or even months. Though their attack they know that they will be free for their regular period. It may be said that this periodicity exists merely in the exciting cause, for instance that supper might be the cause of the nightly attacks, a Sunday dinner, or Saturday night's dissipation, of their occurring once a week, so on. But there are cases in which there can be no such explanation, yet in which there is a distinct tendency to periodicity in the recurrence of the attacks. Again if the recumbent position were the only element in the explanation, it should require a certain amount of time to bring about its result, i.e., a patient was to be a few hours earlier or later, the attack also should be anticipated or delayed for a few hours accordingly. But
But such is not the case, for whether the patient goes to bed at 9 or 12, he is awakened just the same at 2 or 3 in the morning wheezing flattering for breath. Patients have frequently sat up half the night in the hope of waiting off the attack, but it comes on just the same at the usual time. Dr. Hyde Saltis mentions several cases in which the attacks were not gradually increased during sleep, but only came on after the patient has awakened in the morning. He also records a remarkable case of a night porter whose duties kept him up all night, so that he was in the habit of sleeping all day, yet his asthmatic attacks still continued to come on at 5 a.m. in the morning, which has always been their usual time of occurrence. Other facts also point to some inherent periodicity, for in many instances the usual exciting cause is only efficient to produce an attack at about the period when the paroxysm is due. For example in cases in which the usual exciting cause is some hectic fever, some after an attack actual inflammation is harmless, but when the usual interval of freedom has nearly elapsed a slighter error is often sufficient to raise the paroxysm. Now this periodicity is a very curious feature strongly reminds one of a certain class of nervous disease, viz. the paroxysmal neuroses, epilepsy, migraine, paroxysmal

Paroxysmal mania. These are intimately connected, sometimes alternate
with one another. The periodicity of migraine is sometimes
very exact. Trouseau mentions a case in which the
attacks occurred every fortnight almost to an hour.
The remarkable feature is, as has been stated, the
usual exciting cause being only efficient at about
the period when an attack is due, is also eminently
the case in migraine. There are several cases on record
of asthma having been associated, even alternated, with
epilepsy, one form of minor epileptic seizure is statin
epilepsia. These cases however will be fully discussed later.
In this resemblance of asthma to a paroxysmal neurosis,
its occasional connection with epilepsy, we have a striking
suggestion. May not the asthmatic paroxysm be the
analogous of the epileptic fit, or the paroxysmal headache?
And may not the peculiar unconsciousness which constitutes
the asthmatic constitution be a perfectly analogous
condition to that of the subject of epilepsy or migraine?
A condition of instability of certain nerve-cells, which from
time to time discharges themselves, in an explosion,
any little spark being sufficient to excite the discharge
when the unstable cells have reached the limit of their
power of inhibition, or internal resistance, precisely
as occurs in epilepsy or migraine. The sign of nervous
irritability which precedes the attack also favours this view.

2. Gowers, ibid., p. 847.
With regard to the very frequent occurrence of attacks in the early hours of the morning, another element may come into play, for at this time the whole organism is at its lowest state of vitality, the nervous system is in its condition of greatest depression, the inherent inhibition of any unstable nerve-cells at its lowest ebb, so that any slight excitation e.g. a little pulmonary congestion, would suffice to cause their discharge. It may be advanced in favour of this, that this period of the early morning is by far the most frequent for the occurrence of nocturnal emissions, for the same reason, slight stimuli being now sufficient to overcome the diminished inhibition of the nerve-cells.

**The Altered Rhythm of Respiration.**

The altered rhythm of respiration is a constant and conspicuous feature and requires explanation. Normally inspiration is merely slightly shorter than expiration, in the proportion of 6 to 7½ (Sisson) but in the asthmatic paroxysm, while inspiration is very little longer than normal, his sometimes even shorter; expiration is much prolonged, sometimes even as much as 4 or 5 times as long as inspiration. This must be due to one of two causes; either the obstruction to inspiration is less than that to expiration, or the expiratory forces are less able to overcome the obstruction than the inspiratory. It is

is impossible to conceive how any obstacle in the bronchial tubes could oppose expiration more than inspiration, for if movable, one would certainly expect that the obstacle would get drawn into the narrower end of the tube with expiration, so much the latter considerably, whereas any expiratory force would tend to displace it into a wider part of the tube where it would naturally cause less obstruction. Hence it must be that though the obstacle remains the same the expiratory forces are less able to overcome it than the inspiratory. There is no doubt that the forces concerned in inspiration are certainly much more powerful than those of expiration! But as concerns the forces concerned in each case.

During ordinary expiration we have 1. the elasticity of the distended lungs, the chief force 2. the weight of the chest, which tends to pull from the position to which it was raised during inspiration, into its normal position 3. the elasticity of the costal cartilages, which are slightly rotated when the ribs are raised, 4. the elasticity of the tense abdominal muscles, which are stretched forward by the abdominal viscera pressure from the diaphragm during inspiration, ordinary expiration is thus entirely passive and non-muscular.

During forced expiration in addition to the above forces, we have 1. the abdominal muscles which contract to press the abdominal viscera up against the diaphragm.

2. The triangularis sterni, which depresses the sternal end of the lower cartilages ribs from the 9th to the 1st
2. The serratus posterior inferior, which depresses the lowest four ribs, causing the others to follow.
4. The quadratus lumbrorum, which depresses the last rib.

During ordinary inspiration, we have 1. the diaphragm, a
5. The levatores costarum, longi et breves, 3. the
2. The serratus posterior inferior, major et minor, 6. the
4. The sternocostalis, internus and externus intercostales. (Rutherford)

During forced inspiration in addition to the above we have
3. The scaleni 2. The sterno-mastoids 3. The trapezius
5. The pectoralis major et minor, 7. The serratus anterior
6. The rhomboids 3. The external oblique vertebrealis 8. The serratus anterior major.

It is thus evident that the inspiratory forces are
much more powerful than the expiratory, result overcome all obstacles with much less effort. There is another
joint also—the very collapse of the elastic structures in
the lungs, to the chest walls, which takes place in
expiration, would tend to lessen the lumen of the obstructed
tubes still further, especially if the obstruction were due
to spasm or contraction of the muscular fibers in the
tracheal walls. For these fibers could now act unopposed,
Whereas in inspiration they are antagonized by the
resistance of the tubes to expand.

Certainly all the extraordinary muscles of inspiration
are brought into as violent action during expiration.
expiration, all this force would not be necessary to overcome the slighter obstruction to respiration, but it must be remembered that notwithstanding all the patient's efforts, the blood is still imperfectly aerated, the patient endeavours to compensate for the deficient expulsion of carbonic acid by respiring an increased amount of oxygen, hence the inspiratory muscles strive to still further expand the already extended chest.

The Distended Appearance of the Chest.

Why is the chest in an apneumatic paroxysm in a condition of extreme distension of full inspiration, the lungs in a similar condition? I think the explanation is as follows—inspiration being easier than expiration, a little more air passes the obstruction entering the alveoli at each inspiration than the following expiration is able to expel, consequently the lungs get gradually distended to their utmost capacity. The most powerful efforts at expiration are then only sufficient to expel a small amount of air, which the more powerful forces of inspiration are easily able to replace.

The Pulse, Symptoms of the Apneumatic Paroxysm.

The pulse is small, feeble notwithstanding the powerful action of the heart, because owing to the imperfect aeration of the blood there is a sequestration in the lungs, the pulmonary circulation is obstructed, hence less blood passes through into the left side of the heart, while the right
The Sputum.

The peculiar character of the sputum, its almost constant expectoration towards the close of the paroxysm, early attracted attention. Some of the earlier writers, e.g. Bree and Bean, considered it to be the actual cause of the dyspnoea. This theory was supported by Layden in 1872 on his discovery in the sputum of certain angular elongated octahedral crystals, now known as "Charcot's Crystals." He still further inclined to this view on discovering later in the same year, "tubular corkscrew-shaped" bodies with obliquely spiral lines and a narrow homogeneous-looking thread, which ran along their axis terminated with a spiral twist. He considered that these spiral bodies were less concerned in the production of asthma than the crystals, whose sharp points, he thought, irritated the terminal filaments of the vagus, thus producing a bronchial spasm. The spiral structures were later more fully described by Curschmann, hence were named "Curschmann's Spirals." He considered them to be the

The center of a peculiar form of bronchitis, "bronchitis excitation," causing dyspnea resolutely, its being pathognomonic of asthma. The characteristic elements of the sputum consist of little grayish-white mucous pellets, of the consistence of jelly, very iridescent, tenacious. The points in the sputum when the Charcot-Lehman crystals are to be found can often be easily recognized by the naked eye, appearing like small grains of sand. Under the microscope they are seen to be slight bluish shining elongated octahedrals, of great variety in size, adherent by masses of epithelium. They are very often mixed with peculiar fine granulated round cells, which look as if they were filled with dust; together with these are found spire-shaped bodies, slightly glistening, which Kerouvri suggests are a transition-stage of Charcot's crystals. The crystals and the spire-shaped bodies are especially numerous upon the spirals. Some of them are distinctly visible with a simple lens, but they vary so much in size that others are only to be seen with a Hartnack no. 8.

These crystals are insoluble in cold water, alcohol, ether, chloroform, but are readily soluble in alkalies, mineral acids, warm water, ammonia, acetic acid—which murky allays them to mucin, a form which Saltkowskie declared them to be. They are identical with the crystals discovered by Charcot in semen, which Kömpfer has shown.

3. Among Hare, op. cit., pp. 521-522.
shown to be a phosphate of urethanes, also with the crystals found by Neumann in the blood marrow in leukemia.
Friedreich Jæckel found them also in the fibrous plaques of bronchitis, and Bisgoergo von Jæckel found them in bronchial catarrh without asthma, they have been observed also in the tissues in cases of anchylostomiasis. Levey found them in nasal polypi, but more especially in the pale greenish gelatinous masses in patients not affected with asthma. He was unable to discover them in the hypertrophied tissues of tumors encountered in certain cases of asthma. Vierordt states that they are found in acute bronchitis, chronic purulent bronchitis, tuberculosis.

They seem most abundantly during, after the attacks. They are always found in greatest abundance with the spirals, both are the products, thus the cause, of the peculiar bronchial catarrh caused by the stasis, congestion in the bronchial mucous membrane.

In the crystals are a form of mucous, there have been made to develop directly from it in the spirals, in Spectrum protected for several days from evaporation. the spirals exist in greatest number in the earlier stages of the attack, when the crystals may be entirely absent. They exhibit the form of casts of the finest bronchial tubes, hence are true probably to a fibrous catarrh of these tubes. With some practice they are

5. Vierordt. op. cit. p. 179.
recognizable with the naked eye, under a glass cover, when somewhat spread out by very slight pressure, there may be seen a spiral twist, often in the centre a brighter line which is generally narrow. This central line, which can never be made to exhibit a sharply defined contour, which is sometimes entirely absent, is according to Kirschner, not a material structure at all, but merely "an optical image of a space empty, or of a strand of tightly twisted fibres in the centre of the spiral." Dr. Burkard 2 states the latter to be the true explanation. They are especially abundant in asthmatic patients; in many cases, only at the time of the attack, but in some at other times also. — O. Kirschner, Von fakirchen räthes have observed them also in crumorous phenomena. Kirschner has observed them in a case of very chronic tuberculosis of the lungs. According to Pel they consist largely of mucus, and we doubt the product of a peculiar bronchial catarrh. These mucus casts have been very fully described by Dr. Burkard, who considers them to be the product of a peculiar fibrinopellicin inflammation due to a streptococcus, which is the essential cause of asthma; to be pathognomonic of the disease.

This question will be fully discussed under the head of pathotopy. Other peculiar cell-elements in the sputum have been noted. Müller 6 demonstrates numerous large lymphatics

Symptoms

Lungs with pale yellow pigment granules, which show
activity for eosine, were called "eosinophile cells" by Shibata,
who discovered them in bronchitis.\(^1\) They found these same
cells in nasal polyps, especially in the glomerous masses
with but little gland structure.\(^2\) Burkart describes
also, "chains of streptococci, each individual of which
measures about 0.4 μ in size after Gram's method;
+ a small bacillus with round-off poles, slightly
knobly, between 2 & 4 μ in length, about 1 μ broad;
also staining — with the exception of one spot in the
centre — after the method of Gram. Some individuals
appear to be oval, faintly stained in the body but
deeply so at the poles; while others are round, much
smaller, apparently altered by inflammation."

Such are the characteristic constituents of the symptom
of asthma.\(^3\) In some cases these may be streaks of blood
from rupture of congested vessels, or have considerable
hemorrhages. The symptom generally ceases to be
expectorated after the attack is gone, but it may persist
for some days or even weeks after.\(^4\) There is no
doubt that it is the fibrinous exudation formed by a
cataract of the smaller bronchi. This exudation, however,
cannot be the immediate cause of the dyspnea, for the
following reasons. Firstly, any onset a paroxysm comes on
suddenly causes intense dyspnea for a few minutes, other
rapidity subsides, before it would be possible for the

2. op. cit. p. 59.
the exudation to form block up the tubes. Secondly, there is such a small quantity of exudation that it could only block a small number of tubes, and cannot possibly account for the severe dyspnea of an asthmatic paroxysm. And thirdly, the dyspnea has already subsided to a considerable extent before any cough or expectoration takes place. Therefore there must be some other element present to cause this violent dyspnea, though this exudation necessarily helps to diminish the loosen of the tubes and aggravate the condition.

The Tendency to Habitue.
It certainly appears that asthma tends to maintain its individuality in each case, with respect to cause, periodicity, type of the paroxysm. In some patients certain localities only will cause attacks, nothing else having any effect. In others certain objects, and in others the emanations from houses, dogs, or cats, are the sole exciting causes. Each individual maintains his own peculiar susceptibility to the paroxysm, existing in his case, is quite unmixed by any other. So also with the periodicity, the length of the attack. Some patients always have a short, sharp paroxysm in the early morning, others suffer during the day or early part of the night; in some the paroxysm recurs every night, in others only for two or three nights, others leaves a long interval of freedom.
Others again merely suffer from rhinorrhea and slight shortness of breath, regularly every morning about 3 or 4, which keeps them awake for an hour or two, then passes off—yet never suffer from an urgent paroxysm. In many cases, the attack always lasts for 2 or 3 days with mighty exacerbations, then disappears for a period of perhaps weeks or months.

It appears in fact as if a peculiar habit had been established in each case, for the actual pathological condition which is the cause of the dyspnoea is the same in all. Nothing is more striking in this connection than the behaviour of asthmatics to ippecacuanha. Some are quite unaffected by it, in others it will injuriously cause a paroxysm, in others as injuriously relieve it. This tendency to maintain a particular behaviour in each case, again strongly reminds us of epilepsy or migraine. So also the habit may be interrupted or broken off for a time by various circumstances, just as sometimes occurs in epilepsy and all nervous irrevocably of a repetitive type.

**Change of Type.**

"It is very rare," writes H. Salt, "to meet with a case of asthma on which considerable changes do not pass in the course of years. From being irregular, the symptoms have perhaps become confined, from being occasional they have become states, attacks..."
Changes of Type.

"It is very rare," writes Dr. Hyde Salter, "to meet with a case of asthma in which considerable changes do not pass on the course of years. From being irregular, the symptoms have perhaps become confirmed, from being occasional they have become stated; attacks
attacks that were formerly confined to the morning now extend throughout the day; remedies that were formerly inefficacious have now become worthless; a more scrupulous care is necessary in avoiding possible excitants, whose number is greatly increased. It must be remembered that many of these changes are the necessary consequences of the long continuation of the disease, and cannot be ascribed to any inherent tendency to change of type, such as is met with in epilepsy and migraine. There are however some cases in which the attacks, from being rare and severe, change to a frequent mild type, and vice versa. This particular kind of change of type is very common in epilepsy.

Capriciousness.

The utmost contrariety and caprice are exhibited by asthma in almost every particular. In the extraordinariness of the exciting causes; in the effects produced in different cases by the same drug; even in the reaction of the same case to a particular drug; in the influence of various climates; in the abnormalities of respiration over different cases. Here again we are strongly reminded of epilepsy and migraine.

Complications and Sequelae.

It is not surprising that we get distinct pathological changes as the result of oft-repeated attacks of asthma. The severity, the frequency, and the duration of the paroxysms are more important...
important factors in the production of these changes than the mere duration of the disease. For if the attacks are mild, far-between, short, the tissues have ample time to recover before the next attack comes on, consequently maintain their standard of health, especially in young patients. But if the attacks are severe, frequent, prolonged, the tissues suffer severely at each attack, have not time to recover before the next one comes, and hence reach at a lower, broken standard after each attack. Thus it is that if we examine the chest of an asthmatic during an interval, in the former case we find no evidence whatever of any pathological condition of heart or lungs. But in the latter, we find that chronic bronchitis, emphysema, dilatation hypertrophy of the right side of the heart become gradually developed. The air vessels, from their repeated over-inflation, efforts to expel air against an obstacle, gradually lose their elasticity, become over-dilated, permanently emphysematous. The smaller bronchi, never completely recovering from the repeated congestions becoming the seat of chronic congestion and inflammation. During the imperfect circulation of the blood in the lungs, the consequent stasis, of pulmonary obstruction, the right heart becomes over-inflated, hypertrophied to overcome the increased resistance, finally becomes dilated. This condition is still further aggravated by the diminished capillary area, resulting from the emphysema, finally
finally results in chronic venous congestion. The confirmed asthmatic exhibits a very characteristic appearance and physique. He is generally thin, high shouldered, stooping, with a round back. His arms appear to hang loosely at his sides, they are bent at the elbows and are somewhat thrown back. His face is pallid or cyanic; his expression haggard and anxious, his eyes look weary, and are turgid and watery. The cutaneous veins everywhere are too apparent. The lines of the face are deep and well marked, the cheeks hollow, and the mouth generally a little open, with the jaw hanging. The hands are thin, cold, bluish, and generally trembling. The voice is thin, feeble, hoarse. The patient speaks in short, interrupted sentences. He generally has a little short dry cough, but appears to be constantly wheezing short of breath. The configuration of the chest exhibits two distinct types. In confirmed asthma in whom the disease has not begun till after childhood, when respiration was more or less complete, the chest is barrel-shaped, or horse-shoe-shaped, else flat and narrow in front, greatly rounded behind, this configuration being the result of the emphysema, and the raised shoulders and stooping attitude adopted to assist the respiration. But in young asthmatics who have suffered from infancy the type of chest is quite different, and is described as "pigeon breast." The sternum is prominent, particularly at its lower end. The whole lateral region of the chest flattens, or even at some parts concaves. The explanation of this is, that, owing to the obstruction to
To the entrance of air, the lungs are unable to follow the chest wall at each inspiration to the full extent; there is consequently a negative pressure inside the chest. The pressure of the atmosphere acting on the soft, flexible chest wall, presses in its most yielding parts, namely the soft cartilages ribs, the sternum is thus thrown out. In addition, to this, the diaphragm acts overpowerfully in the endeavour to overcome the obstruction to inspiration through the lower ribs cartilages inwards; just as it does acting normally, on the abnormally soft ribs, in hiccups.

**Diagnosis**

Asthmatic paroxysms are generally easily recognized. The suddenness of their onset during the night, without apparent cause; the intensity of the syncope, its laboured character, above all the great difficulty of expiration; the loud, laboured inspirations, rhonchi, the rale in the chest, all unmistakably characterizing the disease. We have further, the longer remission note, the absence of cough, expectoration till the close of the attack; the peculiar character of the history; the absence of fever; other small febrile pulse, to act us. When we further observe the gradual cessation of complete relief, the intervals of complete freedom with no signs of any organic disease, the mental tristesse of the patient, together an asthmatic family history, our diagnosis is certain.
Certain.

There are however other forms of paroxysmal dyspnoea, which may closely simulate asthma; for instance, how these are to be distinguished. Paroxysmal dyspnoea may be caused by occlusion of the larynx, e.g., by struma or laryngeal stricture; by oedema glottidis, impacted by a foreign body, or pressure of a retro-pharyngeal abscess, in all these cases the difficulty lies in inspiration, not expiration. At each inspiration, which is excessively long, stridorous, crowing stertorous sounds are produced at the glottis, pointing unmistakably to the seat of obstruction. Paralysis of the abductors of the glottis produces the same stertorous inspiratory dyspnoea. The laryngoscope is a positive aid in the recognition of these conditions. Obstruction of the trachea or a bronchus, by a foreign body or the pressure of swollen glands, e.g., at the bifurcation of the trachea — o of an aneurism, or syphilitic or carcinomatous neoplasm, may cause paroxysmal dyspnoea, with the chief difficulty in expiration; but in these cases we have physical signs, to the contrary of the condition. Also stertorous sounds are produced at the seat of obstruction. In children by far the most frequent of these is the scrophulous enlargement of the bronchial glands, causing pressure in the bifurcation of the trachea.

Dr. Ernest Smith lays great stress on this point, when he goes so far as to say that "most cases of asthma" in early life are due to direct pressure by swollen glands upon the air-tubes; that the paroxysms of dyspnoea so

"are the ordinary "asthmatic attacks" of young children." Enlarged glands pressing on the trachea or bronchi certainly do cause paroxysms of dyspnoea, which frequently occur at night; but there are not asthma, any more than the dyspnoea caused by the pressure of an aneurism is asthma. The symptoms too are totally different; there is loud respirating stridor and the sound of inspiration, there is a violent spasmodic cough, dullness may often be detected over the seat of the enlarged gland, the appearance of the chest of the patient is quite different; death frequently results after several of these attacks, either suddenly or after a few convulsions. Moreover there is not the least reason to suppose that asthma, scrofula are connected in any way: asthmatic children show no signs of scrofula, nor do scrofulous children suffer from asthma, more frequently than other children. In 125 cases, in only 19 was there any history ofhuman disease, 18. in 87. It would be almost impossible to pick 100 cases of any disease, without finding some history of tuberculosis in at least 8. In Dr. Burdett's 150 cases, the proportion of patients with a history of asthma was exactly the same, 87. Certainly enlarged glands pressing on the bronchi might reflexly produce spasm—just as a human pressing on the vagus has been known to do—and cause symptoms similar to those of asthma; but this cannot be called asthma. (1, p. 190. 2, Op. Cit. p. 410. 3, Op. Cit. p. 95.)
A foreign body in a bronchus may give rise to paroxysmal dyspnoea which may not occur till after an interval of some time: this should therefore be kept in mind. Here again we should have violent paroxysms of coughing, stridulous sounds at the seat of obstruction, rasping sounds caused by the inflammatory exudation set up by the irritation of the foreign body.

Acute Capillary Bronchitis may sometimes cause difficulty, for there is severe dyspnoea present, there is great intensity, and ronchi are heard all over the chest. At first also there is very little expectoration. Then are many points of distinction however: its onset is more gradual, the temperature is considerably raised, there is frequent often paroxysmal cough, in the late stages abundant expectoration of purulent glairy mucus, or mucopurulent. With regard to other patients with chronic bronchitis, dyspnoea, the history, the occurrence of paroxysms of severe dyspnoea, guide us as to whether asthma is present or not. Cardiac dyspnoea sometimes resembles asthma to some extent, but it is generally more shallow and panting than labourious, there are the signs of heart disease. Moreover, the dyspnoea does not set in or subside so suddenly, nor does it come on while the patient is at rest, but shows an intimate relation to exertion. There is not the same amount of rhonchi.
all over the chest. Later, when oedema of the lungs sets in, the dyspnoea may become intense, but it is shallow and superficial, there is profuse watery expectoration.

Dyspnoea from spasm of the diaphragm presents very distinct characteristics; there is a sudden abrupt inspiration, often with straining, then a quick violent expulsive effort. Embolism of the branches of the pulmonary artery causes dyspnoea which affects both inspiration and expiration; there is often expectoration of blood; there are also concurrent signs of heart disease, e.g. embolism elsewhere, not infrequently there is sudden death.

The dyspnoea of chronic anaemia frequently comes on during the night, simulating asthma. Here, however, the appearance of the patient is quite distinct; there are symptoms of characteristic of anaemia present, e.g. vomiting, diarrhoea, pains in the head, twitching of the muscles, disturbance of vision, restlessness, or somnolence, stupor, albumen in the urine.

Diagnostic Value of the Sputum

Charcot's Crystals and Auerbach's Spicules cannot be disregarded as pathognomonic of asthma, as they have been observed in the sputum of acute and chronic bronchitis, tuberculosis, and pneumonia. In a case of chronic bronchitis, dyspnoea however, simulating asthma, their presence would be almost diagnostic, as the above diseases could be excluded very often.
Actiology.

When we consider the various exciting causes of an asthmatic paroxysm, it is evident from their extraordinary contiguity, sudden multiplicity, that there must be a something else which is really the cause efficient, the exciting cause being no more than the spark which produces an explosion, rendered possible only by the chemical constitution of the materials ignited, as Dr. Gowers happily remarks with regard to the actiology of epilepsy. We shall therefore first throw the prevailing conditions, and afterwards the exciting causes.

A. Provoking Conditions.

Herein, there can be no doubt regarding the heredityness of asthma: yet on this point writers are divided. Dr. Gowers remarks, "asthma is said to be hereditary, but I cannot say that it certainly is so in the sense that the asthmatic parent passes on the asthmatic tendency to the child, though this is so sometimes." Dr. F. T. Whistler states that "heredity plays a very insignificant role in the production of asthma." On the other hand, we have the evidence of Dr. Hyge Salter, R. J. Pagge, W. Frederick Taylor, D. Gustave Smith, and others, which is directly opposed to this opinion. Dr. Hyge Salter's statistics are the most complete on this point; out of his 217 cases there was a family history of asthma in 84, or in over 38%. In most of these

2. Amory Hare, op. cit., p. 524.
the heredity is extremely well-marked, the asthma having descended through several generations. Dr. Berkaft, after the rigorous exclusion of all cases in which the ascendants themselves were not under observation, found the hereditary element strongly marked in 16% of his cases. Piegel states that he observed that in cases in which asthma was transmitted from father to children, the disease occurred in these cases at the same age as in the parents, disappeared spontaneously at the same age as in the parents. 2 Dr. Heye Salter calls attention to the fact that sometimes several brothers or sisters in a family are asthmatic, without the parents being so; there are 6 examples of this in his 217 cases. 3 Dr. Walshe also observes the same fact.

Dr. Fagge in his relation remarks, "this kind of connection, which is not hereditary, so much as coincidental, we have already noticed in certain uncertainly nervous maladies, e.g. Thrombosis disease." It may be remarked that this latter disease bears other points of resemblance to asthma. - It is a spastic movement, exhibiting itself in a hyper-irritability of some part of the motor paths so that an ordinary impulse - which in an ordinary person would produce merely an ordinary contraction of a muscle - in a person with this disease produces violent irregular spasmodic contractions. It is also remarkably hereditary, it affects males

males

much more frequently than females.

Age. All writers are agreed that asthma is very
common in childhood, with the exception of Dr. Ennace-
Smith.  The latter states that it is extremely rare in
children: the reason of this, however, is apparent, as he
considers that almost all the cases which are usually
ascertained to asthma, are really merely dyspnoea due
to the presence of enlarged glands in the bifurcation of
the trachea; that there is not the least foundation
for this opinion has already been shown.

The following is Dr. Hyge Salter's table of statistics with regard
to the age at first access of the disease. 2

<table>
<thead>
<tr>
<th>Age Range</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-10</td>
<td>11</td>
</tr>
<tr>
<td>10-20</td>
<td>60</td>
</tr>
<tr>
<td>20-30</td>
<td>30</td>
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<tr>
<td>30-40</td>
<td>44</td>
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<td>40-50</td>
<td>44</td>
</tr>
<tr>
<td>50-60</td>
<td>44</td>
</tr>
<tr>
<td>60-70</td>
<td>44</td>
</tr>
<tr>
<td>70-80</td>
<td>44</td>
</tr>
<tr>
<td>Total</td>
<td>225</td>
</tr>
</tbody>
</table>

Thus in 1/3 of all his cases the first access occurred
during the first decade of life. In one of the cases of access
during the first year, distinct symptoms of asthma
were recognized at 14 days of age, in another at 28 days
in another at 3 months. Thus early infancy is
accessible to asthma, the fact as Dr. Hyge Salter &
Dr. Godwin observe, many of the best-marked sympto-
matic cases begin at this early date.

Dr. Parkart's statistics are equally conclusive: the following is his table of the ages at access:

- Water 10 years - 63
- from 10 to 20 - 88
- 20 to 30 - 37
- 30 to 40 - 23
- 40 to 50 - 15
- 50 to 60 - 7
- 60 to 70 - 3
- Total 180

Thus in his cases also, $\frac{1}{3}$ developed the disease during the first 10 years of life. The earliest ages at access he records are, 5 at 6 months, 1 at 9 months, 13 at 18 months, 15, 7 out of 180 during the first 18 months of life. Hence through there is no time of life at which asthma may not make its appearance, childhood is by far the most prolific; as one would expect in the case of a functional nervous.

Sex. There is no doubt that asthma is very much more common in males than in females. Of 207 of Dr. Hyge Salt's cases, 138 were males and 69 females; a proportion of 2 to 1. Of Dr. Parkart's 180 cases, 101 were males and 79 females. Thus, Hyge Salt's explanation of this fact is that men are more exposed to the weather, to the near stear: hardships, to the stress of violent respiratory efforts, intemperance.

All these conditions would certainly contribute to the formation of the disease, but this does not appear to be the true explanation, for it is in the earlier years of life, before these conditions would have come into effect, that the

that the difference in the incidence on the two sexes is most marked. This is shown both by his own statistics, by those of Dr. Berkart 2 for his own 221 cases only 41.7% of the males were over 30 years of age, while 41.4% of the females. In Dr. Berkart's 180 cases, the difference only existed in a marked degree during the first decade of life, when there were 39 males to 24 females. Certainly male children are more exposed to the rough contacts of life, more than female, and are more liable to disease generally.

Functional nervous derangements, however, are far more common in females than in males; through Thomsen's disease, which is undoubtedly a functional nervous, is an exception to this rule.

The Menopause. In a good many cases of asthma in women the first access occurs at or about the period of the menopause. Of Hyde Salters cases of a total of 70 females, in 22 the first access occurred between the ages of 33 and 50, most of these being between 40 and 50.

Pregnancy. The strain of gestation sometimes proves the starting-point in the case of asthma, as of other diseases.

Climate. Climate has no determining influence; asthma is as common in hot as in cold climates, in summer as in winter. This proves that the weak spot is 1. Op. cit. p. 410 et seq. 2. Op. cit. p. 101.
not in the lungs themselves; for in all this class of case climate has a very important influence, as one would expect.

Race. Asthma is confined to one race: the black races are equally liable to the disease with the white. Social Condition certainly exerts a considerable influence. 

The disease is much more common among the more intellectual classes, especially among those who lead intellectual and sedentary lives, namely, clergymen, solicitors, medical men, & students.

Antecedent Diseases.

Measles, whooping-cough, infantile bronchitis. It has often been observed that an attack of measles, whooping-cough, or infantile bronchitis, appears to be the starting-point of asthmatic attacks. A child has perhaps been perfectly well until it has an attack of measles or whooping-cough; but after that has passed off, it becomes the subject of asthmatic attacks. According to Dr. Hyge Sailer, 80 per cent of cases in the young fail from one or other of these three diseases. In Dr. Berkam's cases the proportion was 54 per cent.

The explanation of this is that while the child is in a high state of health, the asthmatic tendency is held in abeyance; but when he has an attack of measles or whooping-cough or bronchitis, his whole system is very much depressed for a considerable time.

time.
The respiratory system suffers severely in these diseases, as reduces to a low standard of resistance. Hence it is that the time of measles, whooping-cough, infectious bronchitis, is the point of life at which the asthmatic bronchitis most frequently gives the first evidence of its presence; thus being most cases the first occasion of serious depression of the nervous system, the first chance the bronchus has of gaining the ascendancy. After this, each attack renders the patient easier for the succeeding one, and the habit becomes established. So it is also that in just other cases, it attributes to an attack of diphtheria, coma, or acute bronchitis; all of which depress the vitality seriously. Severely inflicting the respiratory system.

Emphysema. The very frequent occurrence of emphysema in asthmatics has led many writers to believe it to be the cause of the asthma. Dr. Reid in 1840 contributed a paper to the Medical-Chirurgical Transactions, "On Emphysema and Asthma"; in which, after contrasting both the necessity of the existence of muscular elements in the bronchial tubes, he states that there are two forms of asthma — one of which is the asthmatic caused by emphysema, the other a spasm, or suspension of the normal action of the muscles of inspiration. Dr. Eustace Smith also supports the view that the emphysema is the cause of the asthma, — "Asthmatic children," he states, "are generally the subjects of emphysema."
Emphysema which gives little evidence of its presence till the lungs are attacked by a fresh cataract, the sequestrates always assume the catarhal form, since the subjects of the disease (asthma) are generally sufferers from emphysema of the lungs, the attack of dyspnea occurs as the consequence of a fresh cataract. In 61 of the cases, however, there is no emphysema until the asthma had existed for some considerable time. Extremely few young patients have emphysema, one of them more frequently, as the disease is of longer duration. The Hoër Salters' cases show this fact in an eminent degree, for in 180 patients in whom he studied the point, emphysema was present in 61, of these only 3 were under 10 years of age, 20 were between 10 and 30, 38 between 30 and 70.

Gout and Rheumatism. It is not surprising to find that such common diseases as gout and rheumatism, from their necessary occasional coincidence in persons who also suffer from asthma, have been regarded as factors in the etiology of the disease. Malaria and syphilis have also been considered in the same light. There is really no evidence whatever to show that they have any connection with asthma, beyond this occasional occurrence.

Neurasthenia. There is no doubt that the neurasthenic temperament is a very powerful predisposing condition in asthma. Asthmatics are almost always of a neurasthenic temperament, many having also a neurasthenic family history. This question however will be more fully discussed under pathology.
Diseases of the Skin. The very frequent association of diseases of the skin—especially of eczema—with asthma, suggests some intimate connection. In 61 out of Dr. Berkant’s 180 cases, some disease of the skin—chiefly eczema—was present. Dr. Eustace Smith also observes this connection, and West even states that he has never known eczema to be very extensive or very long-continued without a marked liability to asthma being associated with it. The two affections, he observes, may alternate; the one subsiding when the other appears, as in the case of a boy of 6 referred to by Caillet, but they may also be coexistent, the cure of one often following the disappearance of the other.

Eczema urticaria have also been observed to coexist, sometimes with asthma. Dr. Berkant mentions two cases in which there was coexistent urticaria all over the body, and two others in which there was one wheal on the arm, twice at the angle of the right scapula. Dr. Price also records a case, and there was observed a case of association of urticaria with hay-fever.

By far the most frequent, however, is eczema; urticaria coming next. Now the connection of these diseases with asthma, though at first sight it appears very curious, in reality is not so. The difficulty, for these skin diseases are very common, are more apt to occur in the ecdysial, that is, especially under the ecdysial skin, which is a manifestation of a nervous constitution. They are all more or less manifestations of a nervous constitution.
frequently dependent on a nervous state; a form of neurasthenia.”

2. Hiccup also frequently occurs in undoubtedly nervous cases; 

3. Turricea in many cases strongly resembles a paroxysmal neuritis, 

4. being one of the attacks in which the “rumor-stomach” may express 

5. itself. Dr. Fowmen describes “a form of recurrent articularia, 

6. which is distinctly and wholly a nervous affection. It occurs 

7. in people whose nervous system has been over-taxed, too 

8. commonly associated with neuralgic affections; often it comes 

9. on with great regularity at a certain fixed time in the day, 

10. sometimes it replaces an attack of neuralgia at the usual 

11. hour.” — Shaw — he states — “met with several cases of this kind 

12. in which the neuralgia did not appear the articularia, 

13. twice versa, the two never appearing together.”

14. Further 

15. points to be observed are, that in all a predisposition, a peculiar 

16. susceptibility, is necessary for their formation; the exciting 

17. causes being as a rule utterly inadequate; that they are 

18. evidence of an unstable condition of nervous system, which is 

19. easily to be upset. Hence is explained the fact that wrong 

20. anxiety are frequent causes of attacks of cymna turricea.

21. The association therefore of these diseases with asthma, is 

22. perfectly analogous to the association of articularia with neuralgia, 

23. epilepsy with migraine or neuralgia. For all are merely 

24. manifestations of a nervous heredity + an unstable condition 

25. of nervous system. This question however will be further 

26. discussed under pathology. This finishes our consideration of the 

27. predisposing conditions; we now pass on to the exciting causes.


B. Exciting Causes.

The extraordinary variety of the exciting causes renders any attempt at classification very unsatisfactory. It will be convenient however, to divide them roughly into direct, local exciting causes, and indirect remote exciting causes.

I. Direct Local Exciting Causes.

Dust. The dust of the streets or the recently swept room, a dusty mill or factory, will always, in many asthmatics, induce a paroxysm. Dr. Hyge Salter mentions the case of a gentleman who did not dare to take a book from his library shelf, as the dust always brought on a paroxysm. He also describes the case of a clergyman—otherwise but a slighter tertian asthma—who never dared to be present at the annual assessment and distribution of blankets to the poor of his parish, as the dust of stuff always brought on a violent fit of asthma.

Feathers. Very many asthmatics are quite unable to sleep on a feather pillow. Dr. Austin Henry was one of these. Dr. Hyge Salter relates a case of a young lady who did not dare to pass a poulterer's shop, as even this was sufficient to excite a paroxysm.

Smoke. The inhalation of a smoky atmosphere, or the fumes of pitch, of an extinguisher candle, will in many patients induce an attack. The fumes of burning sulphur in some cases induce most violent paroxysms which come on immediately, cause intense dyspnoea in a few minutes, then rapidly subside. Even the striking of a sulphur match is often sufficient to produce instant wheezing.

breath. The most violent paroxysm of asthma I ever saw was induced by the patient merely opening the door of a room which was being fumigated with sulphur; yet ten minutes after he had not the slightest sign of wheezing or shortness of breath. Tobacco smoke also will in some cases cause an attack.

**Vegetable Emanations.**

Hay. The pith of certain grasses is a well-known cause of asthma, as well as of hay-fever. Dr. Walshe mentions the "anthriscum satureae." Mr. Hyde Salter, the "marinus stricta," as being most common of these. Watson relates a story of an incredulous person having concealed some hay in a room into which he then brought an asthmatic, with the result that a violent paroxysm was induced. 3 *Ipecacuanha.* The fact that ipecacuanha sometimes gives rise to asthmatic attacks was first noticed by Cullen, who observed that an apothecary’s wife had a paroxysm every time ipecacuanha was powdered in the shop. It is now known to be a very frequent cause. In this case also incredulous persons have resorted to an experiment similar to that related above, of opening a bottle of ipecacuanha in a room unknown to the patient, but with the invariable result of inducing a paroxysm. Indeed, some asthmatics are unable to approach a pint hedge; others fall victims to the fumes of certain flowers eg. roses, lilacs. Ramage relates a case of "an employee in the East India Company who was obliged to give up a lucrative appointment because the smell of tea always developed an attack of asthma." 4

mentions a case of a patient who could not wear mustard in any shape, or hear it near him, so that he did not dare even to apply a mustard plaster.

Animal Emanations.

Among the most curious of the exciting causes of asthmatic attacks are the emanations arising from various animals. Horses. There are many cases in record of persons who have found it completely impossible to live a dense horses on account of the musk-like emanation of myrrh, that proximity was certain to induce. Dr. Hyge Salter relates several remarkable instances of such cats dogs. The emanations from these animals act as excitants perhaps more frequently than those of any other. Many asthmatics cannot sit in the same room with a cat or dog without immediately experiencing severe asthma. Dr. Hyge Salter mentions some very sensitive patients — one felt an attack coming on in an omnibus but could see neither cat nor dog, finally known he encountered a lady opposite had a small pet-terrier officer in her lap. Another found that his attacks had been caused by the coat of a person who had been playing with dogs having been left in the room in which he slept; on its removal the attacks ceased.

Another patient, a boy, was unable to keep rabbits as pets, as the effluvia from their hutches always induced an attack of asthma. Some patients are susceptible to a variety of animals; one was unable to visit the Zoological Gardens; another was susceptible to horses, men, sheep, dogs, rabbits, even mice, which any horse, cattle or dog-show without having such 1. *op. cit.* p. 140. 2. *op. cit.* pp. 140–146. 3. p. 144. 4. p. 146.
Such a vivid paroxysm that he was obliged to leave. In one case this susceptibility to the emanations of animals persisted through three generations; the great-great-grandfather had been a victim to cats, a nephew could never go near a horse, a niece stay in the room with a person who had been in tents. A patient had an attack whenever he went near any guinea-pigs. Another patient was relieved instantly by the proximity of green. Another, a country-clergyman, was so susceptible to the smell of a hare or a hare-skin, that if he passed a pack, he could at once tell whether there was a hare in the bag or not; when a boy, a very severe attack of asthma had been brought on by a jet from firing a hare under a sofa in the room in which he was sitting. The emanations from a fur-house also have been known to excite asthmatic attacks.

Climatic Conditions. Some asthmatics always suffer in hot weather, others in cold; some in dry weather, others in damp. Fog brings on attacks in many; thick weather in some; in others, a change of wind, or a wind from some particular direction. Breathing air in some, relaxing in others; has the same effect. Some patients can't live near the sea, others can't live in the country, some can't live in towns.

Particular Atmospheres. Unappreciable qualities of air. The close vicinity of crowded rooms, churches, railway carriages in many asthmatics infallibly brings on a severe, different houses in the same district, even different rooms in the same house, agree or disagree with some patients in the most inexplicable way. An asthmatic may be perfectly free in one

room of a house, but if he try to sleep in another, will suffer night after night from severe asthma, though there is no apparent difference in the two rooms with regard to ventilation, furniture, or aspect. Some patients cannot enter a kitchen without having a paroxysm. Dr. Walde relates a case in which complete darkness would always excite an attack, and states that he knew several who had found that light would alleviate the dyspnoea. Such was the case with Trousen's whose attacks were always relieved by merely lighting up the dark room at night. 2

Locality. The efficacy of the "genius loci" as an exciting cause of asthma cannot be doubted. No one knows this better than the asthmatic himself, for he very soon discovers that in some places he invariably suffers severely, in other none, others quite independently of any care or careless
ness on his part, or the season of the year, or the weather.

Dr. Highe Salter relates numerous instances,—"in one it always came on an hour or two after going into the country; in another going to Matlock always bought it on; in another, Hampton Court; in another Denmark Hill; in another it was Brighton; in another, a particular house in Matlock." 3

Many patients suffer constantly in the country, and always
per in towns; while others the reverse is the case. Sometimes
it is only certain towns, certain parts of the country that
troubles. As a rule some are quite asthmatic better than
country air. Of all towns, London, strangely enough, with
its fog, smokes, rinks, affords the greatest immunity, from

from asthma. And not only so, but it is the densest and most congested parts of London that appear to be the most beneficial. Mr. Hyde Saltz relates many interesting cases in this connection; his experience was such as to lead him to say, "I believe that 2 or 70 cases of asthma would be cured by going to live in London; indeed I believe a larger percentage than this."

Dense, large, densely populated smoky cities also possess this beneficial influence, e.g., Glasgow, Manchester, Leicester, and Birmingham. Mr. Waltke also observed this fact, relates a case of a man who could only live free of asthma in Seven Dials. Sometimes, however, this is not the case; patients who suffer severely in towns are quite free in the country. Some again are only free at sea, others always suffer severely if anywhere near the sea. There is the greatest eccentricity, contrariety in different cases. Mr. Hyde Saltz quotes an extraordinary example of this, which happened in Dr. Birkett's practice: "At two asthmatics one can only breathe in London, the other, in only breathe in Norwood; if they attempt to go, the one to Norwood, and the other to London, they are stopped in the journey by their asthma and cannot proceed; what is very curious, they are both stopped at the same place — they can neither of them get beyond Camberwell Green, the one in his journey towards Norwood, the other in his journey towards London; then they are stopped there to go back."

As a rule, a locality which is the opposite of the one in which the asthmatic suffers most will be found most beneficial; but this is not always so, the element of caprice always considerably qualifies any general rule. In some cases, it appears that there are only one or two places.
places
in which the patient has an attack; so that a person
though having the asthmatic tendency, might happen to be
located in a place which was favourable, this till strage
without ever having an attack; whereas if he had chance
to live in an unfavourable one he would have been a
sufferer all his life. Here change of air is not
beneficial to the asthmatic as a rule; the suitable
locality must be discovered.

Inflammations of the Air-Passages. In some patients any slight
inflammation in the bronchial tubes always induces asthmatic
attacks; in others inflammation does not appear to have this
power.

II. Direct & Remote Excitig Causes. These we shall divide
into direct causes acting directly through the visceral
Peripheral Nervous Systems, + ii. Central Nervous causes:

i. Reflex through the Visceral & Peripheral Nervous Systems:

Dietetic Errors & Gastric Disturbance. In many patients the least error
in diet, a slight dyspepsia, will inevitably bring on an attack
of asthma. They do not dare to eat a full meal, or a meal of
any description towards the end of the day, others again are
not in the least affected—so far as their asthma is concerned—
by any gastric disturbance whatsoever. So also with alcohol,
some patients suffer invariably after taking alcohol to excess;
whereas in others slight intoxication will effectively keep off
a threatening asthmatic attack. I know an asthmatic, who,
being obliged to spend a few weeks in every year in a part of
the country where he always suffered severely every night,
night, was obliged occasionally to resort to semi-narcotisation, in order to get a night's rest; the informer said that this never failed to produce him a sound night's sleep, that he awoke in the morning breathing perfectly freely and quietly. In some cases it is only certain ingesta that are objectionable. One cannot eat cheese, another meats, another almonds, prunes. One patient cannot drink strong; another can drink weak, clear for trumpery with impunity, but is sure to suffer if he takes port or sherry. Another could eat or drink anything for trumpery, but if he took coffee afterwards he was sure to have an attack. 6. Walsh mentions a patient in whom white fish, another in whom champagne would reefsily bring on a paroxysm. Many asthmatics do not dare to eat any meat towards the end of the day; others are not done later than three or four in the afternoon.

Coughing, in some patients always induces an attack.

6. Hyde Salter gives a good instance — "a patient who, as a rule, relieved his bowels every morning, if the customary relief took place, he retired to bed with an empty rectum; he awoke the next morning quite well; but if he neglected to relieve his bowels, or his efforts to do so were abortive, he was quite sure to be awakened towards morning by his asthma." 3

Uterine Disturbance. Disordered menstruation may sometimes be responsible, as is proved by a case of Dr. Hyde Salter's, in which he put a stop to some paroxysms of asthma, which only occurred at the menstrual period, by relieving the menstrual disorder from which she found the patient suffered. 4.
Parturition. In two cases recorded the patient had had asthma when a child; it had disappeared at marriage entirely, but reappeared at each parturition, that is at that time.

Pregnancy. Dr. Page Blake of Torquay, has a very interesting case, in which the first appearance of the asthma was at the time of conception, the next a month after, and then regularly up to parturition when it ceased immediately or entirely. In this case it is remarkable that there was no morning sickness whatever; as if, as Dr. Hyge Salter remarks, the pulmonary branches of the vasa were affected to the exclusion of the gastric branches; the paroxysms of asthma replaced the ordinary morning sickness. In this case chloroform was found to be the only remedy.

Menopause. As before observed, in some cases the asthma only appears for the first time at the menopause.

Peripheral Nerve Irritation. A very remarkable case of peripheral nerve irritation causing asthma is related by Dr. Chevassu—

"A man of 50 appeared at an insurance office to have his life assured. He states that he was subject to attacks of paroxysmic asthma, if by any chance cold water fell upon his moustach, or his mouth, or any other way became cold. He was not aware of any other cause of the attacks. The paroxysm came on immediately it was very severe. The case was thoroughly investigated with the result that these statements were clearly established to be facts, this life was refused in consequence."

Intra-nasal Disease. The association of asthma with nasal polypi was first observed by writers in the middle ages. Trouseau also long ago recognized the connexion. In 1871, Vottolini reported a case in which he had cured asthma by removing a nasal polypus; the polypus returned and the asthma returned, again to disappear with the extirpation of the polypus. This case secured considerable attention to nasal affections in regard to the causation of asthma. Haensch in 1874 confirmed Vottolini's observation, and considered that the bronchial spasm was set up reflexly by the nasal polypus. L. H. Hacké, Harrison Allen, and Braund confirmed these observations, and the importance of nasal affections as a cause of asthma became greatly exaggerated. Schmigelow even states that they accounted for 30% of the cases; polypois being responsible for 22% and rhinitis for 8%. There is no doubt that intra-nasal disease is sometimes the cause of asthmatic attacks, as in their course of practice, writers are disagreeing, though most of them reluctantly reflect. In this point of view, Macdonald writes: "from clinical observation it appears to me far more rational to assume that asthma in cases of nasal disease as primary or secondary writers are disagreeing, though most of them reluctantly reflect. In this point of view, Macdonald writes: "from clinical observation it appears to me far more rational to assume that asthma in cases of nasal

nasal obstruction, which is a condition nearly always associated with nasal irritation, is due really to obstruction of the nasal respiration; to the respiring air in fact not being warmed, filtered, or moistened in the nose, passing on to the bronchial tubes, there setting up an irritation of its own. This results in contraction of the bronchial tubes from immediate irritation, or rather, we have a bronchial, not a nasal reflex at all. I have seen such symptoms removed simply by restoring nasal respiration in cases where there were no other symptoms of nasal reflex at all, not even sneezing. Indeed it is generally asserted that nasal polypi is associated with asthma; but it is well-known that in outstanding cases of this sort, the nasal mucosa is peculiarly insensitive to irritate stimulation; there are surely not the local conditions in which we should expect a fluid to get reflexes from local irritation."  

This view receives support from several facts: for it is in cases of polypi or inflammation of the respiratory passage proper, i.e., the inferior meatus of the inferior turbinate bones, that asthma most frequently occurs. Also, the removal of polypi from the inferior meatus has cured a case of asthma, while numerous other polypi still remain in the upper nasal passages; yet in these cases—when there is interference with the respiration through the inferior meatus—that the most favorable prognosis with regard to the cure of the asthma by operation, can be given. On the other hand, however, slight nasal irritation often causes asthma, when there is an appreciable obstruction; and many nervous phenomena are

are known to be associated with intra-nasal disease, which can only be reflex, e.g. epilepsy, of which there are on recent untold cases which were cured by intra-nasal treatment, demulsions, cured by the removal of adenoids. Undoubtedly both views are correct, asthma may in one case be produced reflexly by the nasal irritation, in another directly by the irritation of the bronchial mucous membrane by the cold, dry, unfiltred air. Of the various intra-nasal conditions which produce asthma, pharynx is the most frequent, next to this comes rhinitis, then catarrh of the soft palate, tonsillitis, or ulceration of the naso-pharynx.

Laryngeal disease. Irritation about the larynx, e.g. of the interarytenoid folds, has been known to cause asthma. Glasgow records a case in which he had accidentally applied a concentrated solution of carbolic acid to the larynx for some local affection, with the result that the asthma from which the patient had suffered severely for years disappeared never to return. 2

II. Central or Psychical Causes.

Mental emotion. Sudden mental emotion has frequently been known to cause a paroxysm of asthma; as well as to precipitate an existing attack. Dr. Theophilus Thompson records a case in which sudden fear twice brought on severe asthma in a gentleman from his hearing, as he imagined accidently administering an over-dose of belladonna to his wife. 3

to Walsh, also tells of an asthmaetic, who on suddenly discovering that he had forgotten to bring his cigarettes of stramonium belladonna with him, was so alarmed that he was instantly seized with a violent paroxysm. S. H. Salt mentions a case in which he suffered from such a severe attack that he could not move from his chair, a speechless except in monosyllables, till he got such a fright at seeing his sister fall down in a hysterical fit that he was immediately relieved, sprang from his chair, ran down two flights of stairs to procure restoratives, ran up again, his asthma having entirely disappeared. Within an hour however the asthma was as bad as ever. The same writer also describes a case in which the excitement caused by a fire which occurred opposite the patient's house, expelled all symptoms of an unusually severe attack; and another in which anxiety had the same effect. S. Knight relates a story of an asthmatic who was relieved at once as soon as the stakes got high at cards. D. Hyde Salt mentions a case in which removal excitement brought on the attacks; and another in which it would almost invariably cure an existing paroxysm; adding that he has known two in three such cases. D. Walsh also mentions the case of a patient in whom sexual excitement
"in the nick of time" would always ward off an imminent paroxysm. Violent anger has also dispersed an attack.

Another case is recorded of a lady, an enthusiastic musician, who whenever she felt an attack coming on was in the habit of stopping it by the exercise of the emotions she engages in the exercise of her accomplishment. As examples of central causes, Dr. Hyde Salter quotes a case of a boy with acute hydrocephalus who was suddenly seized with a paroxysm of asthma on two occasions before he died, though he had never suffered from asthma before. Yet, the case of a man of 50 an epileptic, whose fits had certain well-known premonitory symptoms occurred fairly regularly about once a fortnight, but on several occasions after experiencing the usual premonitory symptoms he had an asthmatic seizure instead of the expected epileptic fit. The asthmatic paroxysms alternated in this manner with the fits at regular intervals several times, always occurring at the usual period for a fit but at no other time. Several other cases of association and alternation of asthma with epilepsy have been recorded and are fully described under the head of pathology.

A fit of laughing has often been observed to bring on a paroxysm of asthma, but only when the asthma is hanging about the patient. The rationale appears to be as follows: laughter consists of a succession of short expirations followed by a long inspiration, thus series lung

2. 8th ed. p. 43-44.
being repeated again and again; respiration is considerably hindered, with as is shown by the congestion of the face in violent laughter, and the lungs are likewise congested. This congestion, the violent irregular respiration dyspnoea together with the emotion, are sufficient to induce a paroxysm when the asthma is threatening.

Coughing and sneezing may excite a paroxysm in exactly the same way. Sometimes however they may be simply the first reaction of the nasal and laryngeal mucous membrane to some irritant, which afterwards reaches the bronchial mucous membrane and causes spasm.

Dr. Darwin of Kilmarnock has recorded a case in which paroxysms of dyspnoea were apparently due to a neuromatous tumour of the trachea, which was found to be present. Heberden reports one in which the "asthma" was caused by an exostosis of the upper maxillary ventricle pressing on the trachea. Any irritation of a nerve however produces spasm of the muscles supplied by that nerve; irritation of the trachea caused by the pressure of any tumour may set up bronchial spasm and consequent dyspnoea; this however is not asthma.

So also with those attacks of dyspnoea which are due to the irritation caused by the pressure of enlarged glands on the bifurcation of the trachea, which Dr. Stewart Smith ascribes to "asthma." This completes our discussion of the pathology of the disease.

Pathology.

Many theories have from time to time been brought forward regarding the pathology of asthma; even at the present day the question is still undecided. It will perhaps prove useful and interesting to review the different views that have been advanced. The form of dyspnoea which is now known as true asthma was only recognised after the introduction of physical diagnosis. At the beginning of the present century there were many supposed species of asthma, which were soon shown to be merely dyspnoea due to various organic lesions. The first advance in this direction was made by Cornu in 1806. He revived the method of percussion invented by Amelung, by its means was enabled to recognize certain forms of cardiac disease, which were not at all, or but imperfectly known. He rightly regarded that the "asthma" in these cases was merely the dyspnoea which was the consequence of the cardiac lesion.

With the invention of auscultation came further advances. Laennec showed other forms of so-called asthma to be merely symptoms of a "cataarrh" of the lungs, the pathology of which, up to this time was almost unknown. He found however that there were still two forms of paroxysmal dyspnoea to be accounted for, which he termed "asthma with forcible inspiration" and "spasmodic asthma." These, from the paroxysmal character of the attacks, often caused in many cases by trivial circumstances.

2. Berkart, op. cit. pp. 3 et seq.
circumstances, he considered could only be due to a disturbance of innervation, though several causes he thought, contributed as a rule to the result. The question then came to be, of what nature was this disturbance? It was known from the discovery of Reissener in 1808 that the bronchi were provided with muscular fibres - though their function was untested. Miller & Hurlin, with this fact in view, offered the suggestion that spastic contraction of these muscular fibres was the cause of the dyspnea. Laennec considered that the form of asthma which he had termed "asthma with forced respiration," was due to "the vital expansibility of the lungs being increased from a temporary augmentation of the respiratory necessities of the system, caused by some unknown modification of the nerve influence, " the heart of Reissener's discovery led him to conclude that the other form, "spastic asthma," was due to a spasm of the muscular fibres of the bronchi and abdomen, in which he assumed that muscular fibres were also present. 2 Ansell also concluded that the asthmatic paroxysm was due to bronchial spasm caused by an abnormal irritability of the vagus or uterine nerves, a theory which he bases upon the cases he has met with in which "asthma has resulted from the pressure upon those nerves by enlarged bronchial glands, the lungs being otherwise intact. 3 Ramsay likewise in 1835 + Bengoa + before in 1836 states the opinion that asthma was due to a neuritis of the respiratory organs. 4 Hence in: 1. Am an Hen, Op. cit., p. 519. 2. Hurlin Salin, Op. cit., p. 47; et Berkant, Op. cit., p. 7. 3. Berkant, Op. cit., p. 11. 4. An man Hen, Op. cit., p. 519.
however,
the theory of bronchial spasm has been based merely on the
presence of the muscular fibres in the bronchi, though there
was no evidence of their contractility. But in 1840, C.J.B.
Williams, in a series of carefully conducted experiments,
showed conclusively that the bronchi contracted on mechanical,
chemical, electrical stimulation; though he failed to demonstrate
the innervation of the bronchi. For he was unable to see
any alteration in the calibre of the bronchi on irritation
of the vagi, and concluded that "the muscular fibres seem not to be excitable through the nerves of the lungs."! Romberg in 1841 still further established the bronchial spasm
theory; and in 1842, longer confirmed Williams' results, in
the most part, but succeeded further in proving the
innervation of the bronchi. He showed that irritation of the vagi
causes the bronchi to contract, that section of a
tension of the air-vesicles. 2 He saw with a magnifying-
glass narrowing of even the smallest bronchioles, although he
could not observe this in the air-vesicles, he concluded that
since section of the vagi caused distension of the air-vesicles,
therefore they must contain muscular fibres which are
liable to paralysis, were liable also to spasm. Now
homoem came a reaction. Whitrick, after many failures,
at last saw the contraction of the bronchi caused by
irritation of the lungs vagi, but it seemed to him so slight
that it could not be the cause of the dyspnoea. But, Rosenthal
2. "Traité d'Anatomie et de Physiologie des Systèmes Nerveux," Vol.II:
p. 289.
Rünggerling failed to perceive any contraction whatever, this led Bunsen to the conclusion that asthma was in all probability due to certain morbid changes in the blood—changes which induced a spasm of the diaphragm and the muscles of respiration. 2. Wuu 4 also in 1854 adopted the theory of paroxysmal contraction of the diaphragm, in support of which he argued that the enlargement of the lungs in all directions was enough of itself to reject the idea of contraction of the bronchial tubes; for that the lungs should be smaller. Hitzig, however, a tetanus of the diaphragm had not been observed in man; Duchenne had frequently produced it in animals by paralyzing the phrenic nerves, causing some of the symptoms of asthma—extreme dyspnée, distension of the lower half of the thorax, projection of the epigastrium. Diminished frequency of respiration—but with this difference, that death invariably resulted when the irritation of the phrenic was continued for more than a few minutes. Valette, however, afterwards reported a case in which a fatal attack of dyspnée had lasted for several days uninterruptedly, from the symptoms of which he distinctly recognized a tetanus of the diaphragm. Its occurrence in man being thus established, Duchenne concluded that it was the ultimate cause of asthmatic paroxysms. 5

Bonleugh in 1868 further supported this theory. From careful clinical observation, he noticed during the attack the retraction of the lower zone of the chest, the bony-like vibration of the abdominal walls, the almost absolute fixation of the lungs; all of which he states proved spasm of the diaphragm, that spasm of the bronchial tubes under these conditions was "unbearable is not unimaginable." One form of asthma he considered was probably caused by paralysis of the diaphragm.

Opinion now varied for some years between bronchial spasm of the diaphragm. 

2nd in 1869 stated the opinion that contraction of the bronchial tubes was insufficient to account for all the phenomena of asthma, e.g. the dilatation of the air cells, that there must also be contraction of the diaphragm, as well as of the other muscles of respiration.

This opinion was also supported by Lebert in 1873. German See had previously, in 1865, maintained that asthma was a neuritis of the vagus, accompanied by never-produced by organic lesions; consisted of three elements, bronchial respiration, fibrosis of the diaphragm, emphysema. The two forms he considered essential to the disease, the latter only a complication. Subsequently, however, Paul Bézian proved conclusively the bronchial contractility, under stimulation of the lungs, and vagi.

C. Gerlach and McIlvain afterwards confirmed this result. Quite recently, in 1891, Haymow, with the aid of carac and tracheotomy, made experiments on animals in life, and produced the characteristic symptoms of asthma by irritation of the eosophagus. In 1870, Breines, referring to the experiments of Paul Bert, re-established the bronchial spasm theory more firmly than ever. He denies the low level of the diaphragm ascribed by Trante. to B. Hänel as due to extension of the lungs instead to some spasm of the trachea. On the contrary, Trante also, and more recently, Henriksson, concluded that the diaphragm was firmly contracted during the paroxystic, but both held that the contraction was merely part of an altered mechanism of respiration, consequent upon the presence of an obstacle in the air-passage. At first Trante supposed that the pulmonary tissue was the seat of peculiar degenerative changes, which predisposed to a congestion of the mucous membrane; a catarrhal exsudation or other local cause. He later, however, he was inclined to think the congestion might be of vaso-motori origin. This latter opinion was also held by Sir Charles Clark. Weller, in 1872, states that other facts must ameliorate with the bronchial spasm, chiefly stasis formation of blood vessels.

blood-vessels, with stung action and petit evaporation. Regel supported this
view, referring to the experiments of hoen, who demonstrated a reflex laryngoscope in the larynx of irritated salivary nerves; but
strick brought additional support by demonstrating with the laryngoscope acute hyperemia stung action during
the attack, ut the trachea bronchi appear as was visible.
He held known that spasm of the trachea was equally
concerned in the production of the dyspnea! A theory which
had already been suggested but had not received much attention
was that the spasm was caused by a peculiar bronchial
crivation. Before in 1835, was observed that the secretion of
the dyspnea generally coincided with the expectation of a
peculiar kind of spasm—gray in color, very tenacious,
filamentous in shape, twisted into spirals, and united upon
the ultimate ramifications of the bronchi, in which it had
probably stagnated for a time. He considered that the
production cause of the dyespnea was a bronchial spasm,
but that the essence of the disease was a peculiar inflammatory
process, which caused the increased irritability of the bronchial
muscles. This gave rise to these peculiar inflammatory products.
Bemmer however, subsequently discovered that these spirals
were of frequent occurrence in eromous bronchitis, and his theory remained in abeyance till 1872, when regan

with obliquely spiral lines, and a narrow homogeneous-looking thread, which ran along the axis, terminated with a spiral twist. He also observed peculiar elongated octahedral crystals, which had already been noted by Chevreul, Robin, Jöster, and Schreiber, in cases of bronchitis and "cataarrh sore." These crystals were at first supposed to be a mineral compound, but were afterwards found to consist of a peculiar organic base. They had been observed also in normal, in leukemic bone-marrow, in semen; they were therefore concluded that they were derivatives of disintegrating white blood corpuscles. He considered that the crystals were more concerned in the production of asthma than the spirals, which are sharp points irritant the terminal filaments of the vasa, thus producing bronchial spasm. Herbig's facts were confirmed by many observers; but Dencker and Henkel urge against his theory the fact that the crystals occur in cases in which at the time no dyspnea existed. Vringer considered that the dyspnea was caused rather by the spiral formations obstructing the bronchiales, that the assumption of bronchial spasm was unnecessary and unwarranted. These spirals were afterwards studied more fully described by Arndtmann, who thought with Herbig that they were the product of a peculiar form of bronchitis—"bronchitis exsudativa," causing dyspnea reflexly; that asthma was essentially a neurosis of the vasa.

In 1871 Volkmann drew attention to a case in which he had cured asthma by the removal of a nasal polypus. Hachnich 2 in 1874 confirmed this observation, remarking that the bronchial spasm was caused reflexly by the polypus, but only in cases where there was a special predisposition. E. C. Lamb adopted the same view as Hämme. The former described "asthma with foxtail respiration" as "nervous asthma," defining it as a manifestation of a feeling of want of a more complete respiration than the patient enjoys; the pulmonary expansion distinctly taking place with promptitude, completeness, and uniformity, so as to furnish a general foxtail sound on auscultation. 3

Walde described the same thing and the same name of "nervous asthma," believing the dyspnea to depend on marked circulation of the blood, which probably interfered with its ready oxygenation, directly and indirectly. The breathing being restrictively increased in frequency to make up for the deficient amount of oxygen supplied.

Another theory was that offered by Dr. Bree, that the paroxysm was due to an effort to get rid of some irritating matter in the air-tubes, comparable to the sneezing spasmodic contraction of the bladder, that with the discharge of this matter the attack ceased. 4

4. "A practical inquiry into intestinal respiration, distinguishing the species of convulsive asthma."
Others consider that asthma was nothing more than the
sympathy of bronchitis, due to plugging of the tubes with mucus.
Others again attributed it to inflammatory thickening of the mucus
membrane. Among these was M. Beau, who considered asthma
to be a phenomenon of a particular form of bronchitis which he
called "bronchitis à rides infraintes," that the wheezing of the tubes
both depend on the obstruction of the tubes by tenacious inflammatory
products. This view was also held by Cognat, Burleau, and
again, Rostan, identified it with diseases of the heart, large
como, Villeneuve, and Kirchmayr with pulmonary
emphysema. Other pathologists maintained that the essence
of asthma was humoral; that each attack depended on the
development of some specific humoral disturbance, that the
difference between the asthmatic and one who was not so, lay
in the disposition of inspiration to produce this specific
humoral condition. Some assumed that the blood con-
Your text here.
This opinion was also supported by Williams and Romberg. Bergson urges against it that section of the vagi nerve gave rise to dyspnoea when the animal was kept at rest; but his chief objection was, that it was difficult to imagine a paralysis which suddenly came on, and which he thought ought to have been consistent with the origin and subsidence of the dyspnoeal paroxysm.

Mr. Suttace Smith observes, "without expressing any opinion upon the vexed question of the nature of the asthmatic seizure — whether it be a pure nervous (as is commonly held) or not; it is at least curious that in children, whose tendency to nervous spasm of every kind is one of the physiological peculiarities of early life, pure asthma should be an affection so rarely met with. That while general convulsions may be induced by peripheral irritation of nervous degrees of severity, while spasm-like contraction of the glottis may be set up by a trifling laryngeal catarrh — an attack of paroxysmal dyspnoea from spasm-like occlusion of the smaller air-tubes should be a phenomenon of such infrequent occurrence. That it is extremely rare, there can be no doubt."

We have already seen how truly asthmatic is not at all unknown in childhood, and indeed that one-third of all cases occur during the first decade of life. But as Mr. Suttace Smith considers nearly all the cases of so-called asthma in childhood to be merely the dyspnoea due to the pressure of enlarged glands on the bronchi, the explanation of his difference of opinion is apparent.

The theory of bronchial spasm was very ably supported by Dr. Hyge Salter in 1868. His advocacy was based on a life-long experience of the disease, a careful study of 223 cases, his own personal sufferings, and an almost every page he brings forward proofs of the essentially nervous nature of asthma. Most of his statements and arguments are quoted elsewhere in this paper and need not be repeated here. Dr. Berkart dispose of all this mass of evidence by a few arbitrary and quite unfounded statements to the effect that Dr. Hyge Salter's whole conception of the etiology, pathology, and treatment of asthma had been based on the fanciful statements of patients whose friends and relatives were already ill. It is almost impossible to conceive how anyone who has read Dr. Hyge Salter's carefully considered book—every statement in which is supported by clinical observation—can hold this opinion regarding it.

Dr. Grothard—on discussing the question of the pathology of asthma—observes, "There is no doubt that asthmatic children come of a neurotic stock, that the disease is "a nervous strain," to adopt Dr. Edward Jenner's happy term."

Dr. Fagg, though on the whole favoring the spasmodic theory, observes, "On the other hand, it is by no means clear that spasm can account for such considerable narrowing of the tubes as must be present in asthma; or that spasm can be kept up for so long a time as is sometimes required by a prolonged paroxysm of the disease." But the muscular fibers bear quite a considerable proportion to the
Some of the smaller bronchuses, moreover, become hyperoedematous and thickened after repeated attacks. With regard to the latter objection it must be remembered that the congestion and consequent excitation resulting from the spasm aggraves and prolongs the irritation. Keep up the condition. The longer the spasm lasts the more intense is the congestion the greater the amount of excitation. In those short violent paroxysms, one often sees produced in viva by sulphurics, there is pure spasm, no excitation having time to form; the attack subsides very rapidly.

Another theory which has been lately advanced is that the mucous membrane of the tubes becomes very rapidly swollen by a "phlegmonous hyperaemia", or as Velen terms it, "a dilatation of its blood-vessels through the influence of the paro-motor nerves." Dr. Jaggé advances as an argument in favour of this view, the fact that the catarhal form of hay-fever is attended with an obvious swelling of the mucous membrane of the nose. But if this were the case, there should surely also be a free secretion in the bronchial tubes as the result of the hyperaemia or vascular tumefaction, especially in a prolonged attack, instead of the very scanty secretion of asthma. Moreover it is very difficult to imagine such a sudden access to rapid subsidence of this vascular tumefaction, as would be necessary in the short violent paroxysms of asthma one often sees. Finally the action of the insturtes, by still further increasing this vascular dilatation would be to aggravate the dyspnoea.

The inhalation of amyl nitrite very considerably dilates
the vessels, as is seen by the flushed face & breath.
It removes the dyspnoea immediately & completely, for the short
duration of its action. This could not be the case if the
dyspnoea were due to dilatation of the blood-vessels.

Stirch's observation with the laryngeal mirror of the congestion
of the trachea bronchi during an asthmatic attack, has
also been brought forward as an argument in favour of this
theory. But it is not well known that congestion of the lungs,
by a certain amount, is produced by the asthmatic attack,
as a result of the imperfect aeration of the blood. Moreover,
there is marked dyspnoea of the bronchi in even a slight
attack of bronchitis, but this does not produce any dyspnoea
to speak of. Sir Andrew Clark drew attention to the frequent
cocurrence and alternation of asthma with urticaria, & suggests
that the immediate cause of the jet was urticarial swelling
of the bronchial mucous membrane, "both the mucous & tenacious
phlegm being due to a certain neuro-motor disturbance."

A case of this "urticarial asthma" is recorded by Dr. T.D. Bryce!
This was obviously a case in which the urticarial swelling
implanted the bronchial mucous membrane as well as the skin,
causing attacks of dyspnoea exactly similar to those of asthma.

The rash was coincident with the dyspnoea, they subsided
concurrently; the patient had never had asthma before; there was
an asthmatic family history, no neurotic tendency.

Dr. Berkans records four cases of urticaria occurring
concurrently with asthma; 8 Pheronius records a case of
association of urticaria with hay-fever. Now it is well known that urticaria sometimes affects mucous membrane as well as skin, so that the mucous membrane about the throat or trachea is especially liable; when it affects the smaller bronchi also, the swelling produced would have the same effect as spasmodic contraction of the muscular fibres, would cause an exactly similar attack of dyspnoea. This however is not asthma, this is dyspnoea caused by urticaria of the bronchial mucous membrane. The occasional association of urticaria is perfectly natural, as has been before observed, both being manifestations of a neuritic diathesis, of an unstable equilibrium of nervous system. But very many asthmatics never have urticaria, — for one cannot suppose that it is enquired time after time if the bronchial mucous membrane, as more visible anywhere, suffers from urticaria certainly are not generally the subjects of asthma. Eryema is associated with asthma far more frequently than urticaria, yet no one supposes that eryema of the bronchial mucous membrane is the cause of asthma. The suddenness of some attacks of asthma is also opposed to this theory.

A new theory has recently been advanced by Dr. Peacock. His conception of asthma is so totally at variance with all recognised ideas of the disease, that one hardly recognises the description at all. He states that "what is commonly described as bronchial asthma, is an acute, progressive, may almost encrusted form of inflammation."

inflammation which extends from the pharynx upwards to the larynx, is accompanied by a copious exudation. 1 "It is certain," he proceeds, "that no one becomes suddenly, without some warning, subject to peculiar attacks of asthma. In its typical form the disease, before it assumes its distinctive characteristics, is invariably, for an indefinite period, preceded by a series of symptoms which constitute one of its integral parts; namely, a peculiar form of inflammation, which starts in the pharynx, rapidly spreads upwards along the contiguous mucous membrane, but is at first effectively arrested in its downward course by the projecting tissues of the larynx. This inflammation produces seven fits of sneezing and cough, which are taken no notice of, till at last the asthma is fully developed. 2 The earliest manifestations are cough, burning sensations of the soft palate, pain between the shoulders-blisters. There are two forms of the disease,—acute, which is ushered in by shivering or a distinct rigor; the temperature rises generally fluctuates between 101° and 104° F; at the same time there are signs of constitutional disturbance, such as general malaise, frontal headache, weakness, tacking of the limbs, intense pain between the shoulders-blister is early complained of. The first local symptom is itching, or a burning sensation, as if due to the presence of carbonic acid on the nasal surface of the soft palate. The irritation soon extends to the internal ears, to the nose, to the roof of the mouth, to the root of the tongue. Frequently also the eyes are affected, there is a feeling of pressure, as if

1. Aep. 41. 84
2. b. p. 34-36.
as if
grains of sand were in them, then perhaps in a few hours,
often in the course of a day or two, follow violent fits of
sneezing, distressing cough, oppression in the chest. The
hyperaemia with its exquisitely transmitted rapidly spreads
upwards to the nose and conjunctiva, now that the barrier
has been overcome, also downwards to the larynx & bronchi.
Extensive collateral edema accompanies these inflammatory
changes, the eyelids are heavy, as a rule the face is puffy.
In most cases, there are at the same time various
forms of cutaneous eruption. Often the hyperaemia is
attested by a cyanous elevation on the pharynx, towards
anaesthesia, the irritation of the larynx occasionally gives rise
to a peculiar coughing kind of cough which may readily
be mistaken for pertussis, patients from it only in not
being contagious. After frequent recurrences of the disease
it is chiefly localized in the air passages from the trachea
downwards, occasionally however the pulmonic tissue is
seemingly involved in the peculiar changes which affect the
bronchi. There is at all events a marked dulness over
part of the back of the thorax, thus simulating a lobular,
or by the confluence of several lobes, even a lobar pneumonia.
The chronic form is merely devoid for one of the acute it is
distinguished by the constant recurrence of dyspneal paroxysms,
separated by intervals of more or less complete freedom of
respiration. But this freedom is entirely subjective, this
free from a certain of the respirating wants, by means of
which a compensation is established for the exclusion of
of the organ from its junction. For in all such cases, there is evidence of a persistent but imperfect stenosis of several large branches. Presuming the paroxysm often an incessant cough torments them, but makes no impression on the obstacle; the copious purplish expectoration which accompanies it brings no relief. The temperature always, even in the chronic form, rises, and generally fluctuates between 99.5° F. to 104° F. Occasionally there is continual dripping from the nose, and the discharge of saliva may be considerable as to amount to several pints. A peculiar foam somewhat like that produced in singing poultry emanates from the mouth. It is especially marked in the acute form of the disease, is perfectly identical in all cases. Anorexia was occasionally observed in patients with pneumonitis of the lower extremities. Generally there is dulness over the upper lobes. Such is Dr. Berkart's description of the symptoms and clinical history of "bronchial asthma," given in his own words. It is difficult to know where to begin one's criticism; one fact, however, is obvious, that this is not the disease which authorities all authorities have recognized as "asthma." No asthmatic could recognize his disease by this description. Dr. Berkart's "asthmatic" patients appear to have suffered from various forms of disease. The rigor as access, the high temperature, the intense pain between the shoulders-blades, the distressing cough and copious purplish expectoration, the market dulness at parts, suggest acute bronchitis with broncho-pneumonia.

1. p. 40.
2. p. 447.
3. p. 447.
The anomalies so frequently suggest some cardiac trouble, or the persistently raised temperature, dulness of the upper lobes, constant cough appear to indicate pulmonary disease. When we consider the curious symptoms on the larynx, tonsils, tongue, however, with the crowning cough of pertussis, the persistent but insignificant stenosis of several large bronchi, the dripping from the nose, the discharge of several parts of saliva, the odor of burnt feathers emanating from the mouth, we become hopelessly confused. We become a little encouraged, however, when we find that the cases from which D. Berkant derived his knowledge of "asthma" were all, without exception, the subjects of cardiac, pulmonary, or renal disease; sometimes all three together! He himself states that "a broncho-pneumonia in many cases apparently complicates asthma," and "hyperaemia of the alveoli also dilatation of the right ventricle are an almost constant occurrence in asthma"; that "sclerosis of the arteries is frequently seen in the comparatively young"; that "paralysis of the lower extremities occurred in nearly 7% of all cases," that "in a large number of asthmatics, there are in the one or the other infradacicular regions, absolute dulness with increased resistance, bronchial breathing, bronchophony." These statements throw considerable light on the subject, we may now proceed to the consideration of his description of the pathological anatomy of this disease. He calls "bronchial asthma" of the sort which he considers pathognomonic of the disease, he 1. See pp. 44, 71, 73, 141, 144, 163, 164, 176. 2. p. 136. 3. p. 139. 4. p. 141. 5. p. 170.
the

Tinctures, four kinds: 1. greyish-white transparent pellets, after short attacks. 2. Thick, cavitary, exceedingly tenacious masses of greyish-white colour, slightly putrid, from two to four ounces in quantity, when there is associated cataract of large bronchi.
3. Opalescent, perfectly transparent fluid, containing flocculent particles of yellowish-white colour, in the acute form. All yellow, green, or greenish-yellow beliefs, with tendency to confluence, frequently streaked with blood, in long-standing cases."

These are, for the most part, directly due to bronchitis, pulmonary oedema, and partially bronchiectasis. The constituents of these spuia are described at great length. The greyish-white pellets and flocculent particles, "consist of threads of grey a yellowish colour, either straight or bent, or twisted like a corkscrew, varying in length from 1/2 in. to 1 in., in thickness from that of a hair to that of a woolen fibre." "By the side of these are small, perfectly transparent gelatinous bodies, like grains of boiled sago, very tenacious." These may be teased out, and found to consist of "solid cylinders, some of them twisted like a corkscrew, nearly 2 ms. long, others straight, another ½ in. thick, more than 3 ms. long; others consist of a short thick stem, ½ in. long, with bifid or tripedal division, of which one branch is straight and the other twisted, and minutely a cast of a small bronchus. So also solid cylinders, complete casts, may be detected in the catarhal or micro-pulmonary spuia."

On microscopic examination these solid bodies are seen to consist of round cells closely packed together; between the cells is a network of delicate

1. p. 63
2. p. 64
fibres. The casts carry on their surface a mantle of their substance remarkable threads or cords which contrast strongly with their surroundings, by their milky-white colour and their strong reflection of light. Some of these are slightly bent, others are twisted in the most fantastic manner. The transparent pellets and sago-like bodies are almost entirely composed of these threads.

The structure of the threads is as follows: they are made up of strands of fibres. The closeness or looseness of this texture accounts for their varying appearance. Sometimes the outer layers become loose and pull out at the end whereas the core as it were continues to be tightly wound. In that state they represent the central thread found in social, described by Curschmann. In the meshes between the fibres are sometimes seen in various stages of disintegration of pellets with streptococci.

2. Charcot's crystals, chains of streptococci, and small bacilli. With the advance of disintegration when the casts of threads begin to unwind, their contents to escape, these micro-organisms are the first to disappear. Then place is taken by various putrefactive bacteria. Some of the threads are seen on microscopic examination to be slightly twisted or coarsely striated. When unmelted they appear to be sheaths or pseudo-membrane, derived from the alveolar portion of the lungs. They present casts of the impressions of the air-pouches, and some concentric layers of micro-organisms which resemble the streptococci pneumoniae. In the more florid kind of the spita, there are generally large masses of cylindrical epithelium. The epithelium is frequently so closely
closely
packed together as to form casts of the bronchi with their endothelial
epithelium, which may be seen on their surface bounded by fibrous tissues.
Often spindle-shaped epithelium in various stages of degeneration
surrounds the alveolar ends. As a rule they undergo peculiar
changes: the pointe end begins to lengthen, they are
gradually drawn out into thin fibres at the expense of their
bodies, which at last entirely disappear. — This is indeed
an extraordinary process! — "The especially important con-
stituents are the greyish-white transparent fiddle, containing
the threads thereof; these are pathognomonic of asthma;"
not only so, but they are the immediate cause of the asthmatic
paroxysms. — I will give his arguments in his own words —
"that the coughous sputum," — he states — "is concerned
in the production of the asthmatic attacks can hardly be doubted."
"Notwithstanding the fact that, unhappily interpreted they indicate
only a form of fibrinous bronchitis. — Notwithstanding that
they are found in pneumonia bronchitis where no dyspneal
paroxysms are present, or that occasionally more are found
in the sputa of typical cases of asthma, — further that the
sputa as well as the stethonic signs which they produce,
persist during the intervals, in which respiration is almost
entirely unimpaired, though their presence need not be
infrequent is often met accompanied by dyspnea; still the
affection of which they are a symptom, leads as a rule,
to a transient stenosis of the larger tubes. Hence if they are
met with in connection with attacks of asthma, there can be
little doubt that they are concerned in the production of
"the functional derangement."—This is certainly most remarkable reasoning. — "That the symptoms," he proceeds, "is undoubtedly caused by a transient stenosis of the larger branches, the symptoms of physical signs afford ample evidence; while this is true to a movable pathological process, a fibrous exudation, or the products of its progressive metamorphosis."

In examining the evidence, however, which induced Dr. Pekart to form his opinions, we find that his whole conception of the pathological anatomy and pathology of asthma is founded on the post-mortem examination of one single case, which affords no information whatever on the pathology of asthma, as it is rendered quite inconclusive by long-standing complications of severe bronchitis and heart-disease.

The peculiar characteristics of asthma in patients generally, the pathological anomalies found in this one case form the whole basis of Dr. Pekart's theories.

"The results," he writes—"of the antiphlogistics that have been made and published up to within the last 30 years, have been collected by Thring; but if they have any value they merely show that a form of paroxysmal asthma may occur in connection with all conceivable diseases. Since that time only five such examinations have been reported."

1. p. 149.

2. p. 70.
aged 70, who had from earliest infancy been subject to frequent attacks of asthma. Her constitution had been undermined by care & anxiety. She was also in the highest degree addicted to the abuse of morphia. The case was complicated by dyspnea & albuminuria, which indicated the near approach of death. The lungs were emphysematous; the sputum was very viscid, of a grayish-white color, opalescent, & contained almost always long whitish fibrinous threads, but very few greenish-globular masses. Death took place in a paroxysm attack with cyanosis & delirium. The autopsy performed showed after death showed extensive vesicular emphysema. Both lungs were very large, overinflated & pale, their margins were partially covered with large bullae. The bronchi were not dilated, their mucous membrane was red & not materially affected. The bronchioli contained small quantities of mucus, greenish-pulvinate substances could be squeezed from the cut surface of the lungs. A number of dilated alveoli were found filled with granular masses, layers of a coarsely granular apparently amorphous substance, which considerably reduced their calibers, firmly adhering to the surfaces of the mucous membrane of the bronchioli. In one bronchiolus the lumen was annular, with only a narrow slit in the centre; in another it had somewhat the shape of a corkscrew, leaving a semicircular passage. The state of the heart was not mentioned. What light this case throws on the pathology of asthma is not pointed out, except the case is not further mentioned.
This brings us to Dr. Burkart's own case, which completes the list of those in which autopsies have been made—"Mrs C., aged 39, with a history of pleurisy on her father's side, had had a marked disposition to edema, which were specially severe in the early summer. Fourteen years ago she had been seized with an attack of hemolimtes, in consequence of which she had been confined to bed for several weeks. Recovery had been but imperfect; her breathing had remained embarrassd; soon after she had become subject to typical attacks of asthma. The hemolimtes had frequently returned, lasting each time for several weeks, during which she had been unable to leave her bed. Early in June, 1886, her legs had begun to swell. She went to hospital. Her urine was diminished in quantity and contained albumen. The heart's action was disturbed; stethic sounds were heard all over the chest. She had several attacks of asthma, which were but slightly relieved by foments of burning salt and other stimulants. She left hospital on Jan. 6th, 1887, the edema being considerably reduced, but the tympanal paroxysms persisting in intensity and frequency. On Jan. 14th, 1887, she complained of dyspnea, cough, insomnia, diarrhea, weakness, there were with small stature, fairly good nourishment, pocket-shaped head, pterygium of face and nasoarea of the legs; the thorax was flat narrow, there was flattening of the spine. The respirations were 30 per minute; there was inverse action of the diaphragm; the resonance on both sides was short, high-pitched; there were sonorous tubular sounds.
all over the chest. The pulse was 100, small, soft, regular; there was epigastie pulsation; the carotid impulse was distinct in the 5th interspace below the sternal line, the carotid sounds were loud and clear. The urine was of low specific gravity; there was no albumen. The urination amounted to about 4 ounces in 24 hours; was of grayish-white color, very acrid, foetid, containing all the elements previously described. She was seen again on Jan. 29th, 1887. The cough had been slightly improved, but during the last two days, all the symptoms had grown worse; she has also passed less water. The swelling of the legs had increased; the cyanosis was very considerable. Her hands were cold; there was no pulse felt at the wrist. The systole was very great. Resonance on both sides was wanting; that of complete silence on the right, but rhonchi were heard on the left. The carotid impulse could neither be seen nor felt; the carotid sounds were very feeble. Painful stimulants had no effect; she died next day at 1 a.m. The post-mortem examination was held 24 hours after death. There was some considerable, inexplicable dilatation of the heart. The lungs were not collapsed; were dark brown, rather dry, studded with tufts of fat. Effusion of the right apex; there was collapse fixation of the right base. In the right main bronchus was found a spongy, hooting cylinder of a dark brown color, dry and somewhat friable, consisting chiefly of cylindrical epithelium in various stages of degeneration; here and there were found the milky-white cords. The mucous membrane was almost entirely
entirely

Tumors of epithelium, these were colonies of staphylococci present.

The left main bronchus contained a small quantity of greenish, soft mucus; in one of its divisions there was also a cylindrical formation like the above. The smaller bronchi were dilated, a great many were partially occluded by excrescence which was almost entirely made up of colonies of streptococci; their walls were entirely formed of epithelium.

Both lungs were emphysematous, the bronchi of medium size showed irregular dilatations; in some there was considerable hyperplasia of the mucous membrane, most of them were more or less occluded by masses of detritus. Some of the abscesses were nearly filled with a fibrinous exudation.

There was some ascites. The liver, spleen, kidneys were large, pale, dry, and showed signs of amyloid degeneration. Such is the case on which Dr. Buck has formulated his theory of the pathological processes of asthma; in fact he goes so far as to state that "the clinical phenomena of the chronic form of the disease, could not possibly arise in circumstances materially different from those here described." It is evident that this case was so complicated by severe long standing pulmonary and cardiac disease, as to be even more inconclusive, tapping even less information regarding the pathology of asthma than any of those previously mentioned.

From the study of the above pathological conditions, he deduces the essential changes of the respiratory surface as follows:

"The epithelium first of all exfoliates, in the smaller bronchi it is then gradually drawn out into fibres. This elongation is
is probably effected by the breaking of the heavier body of the cell, which lies upon its muscular portion. In this way the fibres on opposite sides of the walls meet, interlace, from some of the threads previously described. This remarkable explanation is quite beyond criticism; it is simply almost... The desquamation of the epithelium is followed by a continuous emigration of leucocytes, which might so considerably as to completely occlude tubes not exceeding 5 mm. in diameter, be in contact with the fibrin-plastic substances which come at this time, the white corpuscles disintegrate. Most of them are converted into fibrinous, rhythmic threads, while the meshes which they form enclose these cells that resist the dissolution. Together with the degenerated epithelium they compose the larger laminae of the corium. The subsequent metastases of the leucocytes is determined by the anatomical condition of the surface upon which the excision takes place. In the absence of the bronchiae they retain their hyaline character; bone, into the structure of the preservative membrane of the corium. In the larger bronchiae, however, they are subject to the solvent influence of the secretion of the mucous glands, so that the mean the more the less their origin is obliterated. The softer excised margin may be expected, if the vis a torna is sufficient for the purpose. But if on account of its tenacity, it is retained within the air-passage, it becomes the matrix of certain mucous organisms, which by their specific action, contains the softening cement is into greenish, clear viscous masses."

The nature of the parasymp and the expanis as follows: "There can
be little doubt, that the immediate cause of the dyspnoea paroxysm is a transient stenosis of the main bronchi, or one of the branches. 

So more of pneumonia resembles in many respects an embolism; the embolus is harmless so to say, at its place of origin, as only a limited number of tubes is obstructed. Gradually however the embolus shrinks, engenders other regenerative changes, in consequence of which it becomes detached from its matrix and rendered movable. Air can now pass between it and bronchial wall. The vis a tergo thus created, though insufficient to displace it, is too slight to effect its expulsion. Being at its great tenacity the plug is in its onward course arrested, though small it may be in size, it necessarily diminishes the calibre of even a large bronchus. It is impossible to conceive how this could occur; for if the tenacious embolus can be displaced from the small bronchus by a very slight vis a tergo, surely in all reason it should infinitely more readily expelit from the trachea large bronchi by a much more powerful vis a tergo; embolus would only be possible in the opposite direction.

The physical signs moreover are quite inconsistent with any such condition of affairs. — All the symptoms, he proceeds, — of the asthmatic paroxysm, become thus intelligible. The subjective dyspnoea, the forcible respiratory movements are chiefly the consequence of the deficient ventilation of the lungs; the diminished frequency of respiration results from the obstacle within the air passages. Both inspiration and expiration are impeded, but the plug, coming as it does from below upwards, acts in the manner of an expiratory valve, allowing the access but
but presenting the expre of air.” — This theory again is offered as a reason, for the plug would get into a wider channel the further it was driven out, and not possibly act as an expiratory valve till it reached the glottis; whereas the face of quantity reach inspiration would tend to draw it into a narrower channel, where it would act as an inspiratory valve, preventing the access but allowing the escape of air. The mode of cessation of the paroxysms is explained as follows. Although the expiratory trachea fails in supplying the requisite quantity of air, it has nevertheless a certain salutary effect. By enlarging the thorax it rarefies the stagnant air behind the obstacle; thus leads to an increased elevation of serous from the blood vessels in the bronchial mucous membrane. There is consequently a hystops ex nuaco, as it were, which, by the flushing of the tube serves to remove the obstacle, which has hitherto resisted all mechanical pressure. In this way the serousness of the air passages may be fully restored, if all the evacuation is expectorated, recovery is complete. But if the process continues in forming fresh evacuation, or the plug or a portion of it has only subsided to the centre towards the periphery; with the return of the plug to the centre, the asthmatic paroxysm also returns, this alternation of symptoms persists till the chest is entirely cleared.” — All these statements are quite coincident with the physical signs and clinical history of the disease.

With regard to the etiology, he considers the exciting cause of the peculiar cryptopetions inflammation which he terms
Terms brochial asthma was to be the streptococcus found in the exudation. Previously mentioned. — "Notwithstanding the fact," he observes, "that this streptococcus is normally found in the mouth; that it occurs in contagious exudations in measles, whooping-cough, bronchitis, notwithstanding the failures which attended my numerous attempts to satisfy as far as possible the postulates of bacteriological science, in order to arrive at some decision on the point, that the mere presence of an organism known to cause injury, absolutely nothing as regards its pathogenic nature; — still in other cases but a micro-organism can possibly produce asthma, therefore it must be the streptococcus which is most constantly found."

It is unnecessary to discuss Dr. Berkars' views any further; throughout his book all is mere statement of opinion; there is no argument, no attempt at proof of any of his statements. All the observations and clinical experience of others, which militate against his theories, are either simply ignored or arbitrarily denied. His views are inconsistent with the physical signs of the disease, all clinical experience, and are unsupported in any way whatever.

We have now discussed all the theories that have from time to time been brought forward regarding the pathologic asthma, and maintain that the only one which satisfactorily accounts for all the phenomena of the disease is the bronchial spasm theory. As a basis for the consideration of this question, I advance the following propositions: — 1. That true asthma is essentially neurotic, that it is of the nature of a paroxysmal fit. p. 112. 2. p. 110. 3. p. 109-110. 4. p. 65, 68, 113, 114, 121.
paroxysmal

neurons, analogous to epilepsy + migraine, implying unstable nerve-cells of easy explosion. 2. that there is in every case a peculiar trait to present, which consists in instability of these nerve-cells and constitutes the "asthmatic tendency." That given this trait is a trifling cause may be sufficient to overcome the stability of these cells and cause an asthmatic paroxysm; but that without it asthma is never present. 3. that the asthmatic paroxysm is the result of the upsetting of the equilibrium of these cells, is immediately due to a spasmodic contraction of the muscular fibres of the smaller branches. 4. that this spasmodic contraction soon brings about congestion + irritation which aggravates the condition; that the excitation is fibrillary in character, of some casts of the bronchiolar mucous secretions, at the end of the paroxysm, constitutes the peculiar "asthmatic spuittus." That asthma is essentially neuritic has already many times been pointed out—the almost invariably neuritic temperament of the patient; the frequent neuritic family history; the marked heredity; the various premonitory paroxysms; the remarkable organic + mental inadequacy of the exciting causes; the effect of sudden mental emotion in instantly inducing a paroxysm, as instantly dispelling an existing one; the instantaneousness of some attacks, their rapid subsidence leaving no signs whatever behind them; the occasional asthmatic association of asthma with other neuroses, eg epilepsy + tic-tacorio; + the effect of certain
certain remedies which act entirely on the nervous system, in instantly expelling an attack,—all from unmistakeably to a neuron.

There are many points which ally asthma with the paroxysmal neuroses, epilepsy, migraine—which have already been noting; the preliminary paroxysms followed by the symptom interchange, then the interval of complete remission till the next period approaches. The periodicity, the tendency to diurnal tide, the marked periodicity, the neurotic temperament, the capriciousness latter inadequacy of the exciting causes. The occasional association of alternation of asthma with epilepsy, the facts that one form of minor epileptic seizure consists in attacks of acute dyspnoea, are very strongly suggestive, though can only have one ex-
planation. The frequent limitation of asthmatic attacks to the night time is now explicable, for in many cases of epilepsy the fits occur only at night. Several cases of the coincidence of alternation of asthma with epilepsy have been recorded. Dr. Hydr. Salter relates one of a man of about 50, subject to epilepsy, whose fits had certain well known premonitory symptoms which occurred with tolerable regularity about once a fortnight. On one occasion after experiencing the usual premonitory symptoms, instead of a fit following, violent dyspnoea came on, which was characteristic asthmatic dyspnoea, within a few hours this went off, slept him as usual. At the expiration of the accustomed interval after this attack the ordinary premonitory symptoms (the ordinary epileptic fit) occurred. This on several occasions after the epileptic seizure was supplanted by the asthmatic. The lungs were perfectly

2. Gowers. "p. 44.
health before the attacks, the character of the dyspnoea was distinctly asthmatic, each attack of asthma occurred at the usual epileptic period, was preceded by the usual epileptic aura. D. D. Beattie records two cases: one in a man of 50 who had had asthma for 20 years; the first attack of epilepsy coming on at the moment of his consulting a medical man; the other in a man of 50 who had been asthmatic from infancy; lately he had also had occasional attacks of mania as well as of epilepsy. Dr. Lloyd Francis and F. R. P. Taylor also record a case of an infant in whom epileptic fits were replaced by asthmatic attacks. The patient was a woman aged 31, an idiot. At first the epileptic fits occurred twice or thrice a month, but afterwards increased up to 10 a month. Her general health was good. There was no history of cough or other respiratory trouble. She went to bed in the evening of a day on which a fit was due, perfectly well, but soon afterwards was found to be suffering from a typical asthmatic attack, instead of the epileptic fit which was expected. This attack lasted with occasional brief remissions, but never with complete remission for 57 hours; the then had a mortal attack, very well-marked epileptic fit, the asthma rapidly subsided. These cases at least prove that instability may affect the motor pulmonary cells in the respiratory centre, so that the disturbance of their equilibrium may result in spasmodic contraction of the bronchial muscular fibres, causing many of the others, which may persist for a long period.

period,

In spasm of the bronchi is more than probably the cause of the paroxysms of dyspnoea. There can be no doubt as to asthma being a nervous, the question simply comes to be, are the attacks of dyspnoea due to spasm or contraction of the bronchi, or to a nervous disturbance causing vascular turgescence of the mucous membrane. There are many facts opposed to the latter supposition, as has been before observed — to recapitulate them, this vascular turgescence could not come into existence so instantly and subsist so rapidly as it would have to do in some attacks of asthma; moreover, it should cause far more excitation, respiration than takes place in asthma, especially in prolonged attacks. Also the action of minutes would be to dilate the vessels, aggravate the condition. The only real reasons for supposing that mucous swelling of the mucous membrane is the cause, and the occasional association of asthma with urticaria, the fact that such swelling produces exactly similar dyspnoea. But this occasional association has already been explained to be perfectly natural on other grounds.

As to the seat of the disturbance — whether central, or consisting in a hyperasthema of the vagus, — many facts point to the former being the case; e.g., the effect of mental emotion in exciting or intensifying an attack, the production of attack reflexly by remote peripheral irritation, the fact of asthmatic sneezes, sometimes replacing epileptic fits. The strict limitation as a rule, to the respiratory cells of the centre of the vagus
centre of the vagus occurs in is no more remarkable than many forms of minor epileptic
ticurie. And moreover cases have been reported in which
the asthmatic symptoms have been slighter + unimportant,
whereas there may very severe gastric symptoms; the two have
sometimes alternated, there being at one time an asthmatic
seizure + at another a minor attack of colic or distressing.
D. Hyge Sadek records three very striking examples of this.
Hence both the gastric respiratory cells of the vagus may
be affected. On this supposition also the intimate relation
between gastric distress + asthma, which is so frequently
observed, is readily understood. On the other hand if the
condition were actvent but consisted in hyperesthesia of the
vagus, the effect of emotion + minute peripheral irritation
in some cases + the association with epilepsy would be difficult
to explain. Hence if there were hyperesthesia of the vagus
asthmatics should be hypersensitive to all irritating matters
in the atmosphere + the inhalation of irritating particles
should be by far the most frequent cause of attacks,
but this is not so; many asthmatics can breathe distinctly
irritating atmospheres without having any asthmatic symptoms.
Further, irritation of a hyperesthetic vagus should cause
violent crouping as well as spasmodic contraction of the
bronchi. The state of affairs is probably similar to that which
exists in the other paroxysmal neuroses. There is a condition of
unstable equilibrium peculiar nerve cells, in each individual case
there is some special apparent impulse which has the power
to upset this equilibrium, though many far more powerful
powerful
stimuli may be powerless. A precisely similar state of affairs
exists good in migraine, epilepsy, etc. This completes our
study of the pathology of the disease. We now pass on to the prognosis.

**Prognosis**

The prognosis of asthma can be stated with great definiteness. Mostly, with regard to the existence of the disease — if the attacks come on only in certain localities, the patient's circumstances permit him to just a favourable locality, and thenceforth, his asthma will probably never return. For it is certain that asthmatics can live for years in certain localities without the slightest symptom. So also if some definite cause for the attacks can be discovered, this can be avoided, e.g. late summers, alcohol, etc. proximity. If dogs or cats, etc., the patient may never suffer again, or only at rare intervals.

If, however, all localities appear to be the same, the attacks may occur from indefinite causes, or the patient's circumstances do not permit him seeking a more favourable place of residence — age now comes in as a very important factor in the prognosis. If the patient is young, with suitable care treatment, the asthma may often subside at puberty. The chances of the cessation of the disease diminish with advancing years, after recovery very seldom occurs.

Secondly as to the duration of life. Here we are guided in our prognosis by the presence or absence of organic disease of the heart or lungs; the frequency, severity, & duration of the attacks; the completeness of the recovery in the intervals; the tendency of the paroxysms to become more frequent, severe, or otherwise;
Finally the duration of the disease. If there is no organic
disease of heart or lungs; if the attacks are not frequent, are
rarely of short duration, there is complete recovery during
the intervals; if they tend to be less frequent, less severe, life will probably not be shortened; a favorable
prognosis may be given. If however the disease is of long-
standing, there is disease of heart or lungs, the attacks
are severe, prolonged, frequently repeated, no complete
recovery between them is impossible; the patient will
certainly go from bad to worse, this life will be considerably
shortened. The completeness of the recovery between the
attacks is a point of vital importance to be ascer-
tained; for if the attacks are so frequent prolonged that
the congested capillaries, distended air-vessels have not
sufficient time to recover themselves before the next attack,
the congestion + extension become chronic. Bronchitis +
emphysema are increased, life is shortened into risks
increased. Hence chronic cough + expectoration between the
attacks, + persistent wheezing, + shortness of breath, + recent
bronchitis + congestion + inflammation, commencing emphysema,
+ must be considered of grave omen. Another point of
importance is the age of the patient at the onset of the
disease: if the asthma but not commence till after 40,
the prognosis is much less favorable than if it has began
earlier in life, for after 40 the its tendency is generally
towards progressive severity, whereas earlier in life it tends
towards recovery.
In cases of asthma caused by intra-nasal disease, as regards adenoids, hypertrophy of the inferior turbinate bones, deviations of the septum, the progress as to the result of their removal by operation, especially in children, is very favourable. But as regards polyps it is far from being so. Out of 28 cases of Dr. Macmillan's, which were operated upon for the cure of asthma, 12 were manifestly benefited, 8 being practically cured; of these 12, 2 were cases of deviations of the septum, 4 of hypertrophy of the inferior turbinate bones, 2 of polyps, 1 of post-nasal adenoid, the remaining 10 were cases of polyps there were not benefited at all.

The mere length of duration of the disease is not a sole a very important factor in the prognosis as to the duration of life; in one case which has only existed for 5 years might be in an infinitely worse condition than another which had existed for 20.

Simple uncomplicated asthma does not appear to shorten life. In two of Mr. Hyde Salt's cases, the asthma had lasted for 64 years, having begun in one at the age of 3, in the other at 6; in 27 it had lasted for upwards of 20 years — in 2 for 34 years, in 2 for 35 years, in 1 for 37, in 1 for 39, in 1 for 43, in 2 for 45, in 1 for 53 years.

Hastily as regards a fatal termination to the paroxysms, this may be said to be uncommon.

There is not the least evidence to show that asthma tends to develop features as has been stated by some writers.

Treatment.

It is evident that in the treatment of a case of asthma, the first thing to do is to endeavor to find out the exciting cause of the attacks. If this can be discovered, it can be avoided, no further treatment will be necessary. Hence where possible always try the effect of a different atmosphere; for it may be merely town-air, or sea-air, or country-air which disagrees with the patient. So also make sure that gastric disturbances is not at the root of the matter; or disease of the naso-pharynx, the cure of which may at once dispose all symptoms of asthma. When however we have eliminated such causes as these, our endeavor must be to diminish the tendency to the attacks, by constitutional preventive treatment; to shorten and allay the paroxysms when they occur. The former is of infinitely the greater importance, especially in children; for the asthmatic paroxysm is merely analogous to the fit of epilepsy, or the wheals of urticaria.

Constitutional & Preventive Treatment.

Here I offer a most emphatic protest against the usual treatment of asthmatic children; they are kept wrapped up indoors, wore protected against the slightest wind or cold weather by their layers of clothing; are only allowed to go out in warm weather, they are debarred from all healthy games, exercises, for fear running should make them short of breath during an attack. The natural consequence is that they become more and more "delicate", more and more susceptible to trivial
exercising causes; for they are rather likely chance of becoming more vigorous than asthmatic children.

The very opposite would be the method of treatment: asthmatic children should be toned up and invigorated in every possible manner. Their nervous system is already too sensitive. It requires strengthening rather than pampering and sheltering from everything likely to increase its resisting power. Healthy games regardless of all sorts are the best thing possible for an asthmatic child. It should be encouraged in every way. The child should be allowed to play exactly like other children, but care being taken to avoid colds or a trip to the house. In this way his general health and nervous system will gradually become more robust, less susceptible to taxing external causes.

Cold baths too are of great value; should always be used where possible, instead of the over-eating, warm ones to which the asthmatic child is usually limited. For fear the cold water should cause an attack.

As an additional a nerve tonic will be quite useful, especially arsenic solution of iron, with a scintilla or two to diminish irritability, if an attack is threatening.

Dr. Freemond recommends big. Arsenac. M.XX + 1/4 2 Troy. Drachm. M.X XX at bedtime; e.g., for a child of 6. Chloroform 9 to 6 S substaneously at bedtime is also spoken highly of.

Professor Chamber's strongly recommends big. Arsenac. M.X with breakfast tea; it corrects bronchitis to be maintained with

with

Stramonium at nights. The amonic should be continued for

courses of 3 months at a time. B. Cestari finds

3-weeks courses of arsenic the most useful during a
period of several months, with intervals also of 3 weeks.

Strict attention to diet is of great importance; the

child should be fed with the greatest regularity and

plain nourishing digestive food. Eating just before

the stomach should always be prevented. A full meal

should never be allowed towards bedtime, nor reduction in the quantity

drinks.  An amendment in the quantity

of amoniacal muriatic acid should be observed.

By these means the child is rendered more vigorous, his

sensibility to slight external stimuli diminishes, his

intestinal resistance increases, the "grows out of this asthma,

in the case of other patients also a great deal may

be done by this line of treatment;—rigorous bodily exercise

cold baths, careful attention to their regulation of the bowels

are often of the greatest value. Tonics also may be

given, especially arsenic, iron, tannin.

As a preventative, Siroche of Potassium is often of extreme

value, it should always be given a trial. It is best

given in doses of gr v. vili three times a day in the

intervals between the attacks, in many cases effectively

prevents their recurrence. Its effect sometimes does not appear

for a week or two after the administration has been

begun. Bromides also in some cases are beneficial,

but the spirits are of all things the most often successful.

Many asthmatics have themselves found out the efficacy

of vigorous bodily exercise in keeping off attacks, and

that a half day's exercise would bring them immunity.

community
from the expecter attack at night. Dr. Hyde Salte relates
several striking instances of cases met with two or three
The exercise must not be sudden, violent, but prolonged
sustained. Ordinary gentle exercise is not sufficient,
it must be to the extent of absolute fatigue. e.g., a walk
of 20 miles, or a pull in a boat for 4 or 5 miles, a
sustained gymnastic exercise. I have known patients
who found a hard evening's dancing to have an equally
beneficial result, other than understanding the
irregularities attendant on suffer etc. Dr. Hyde Salte
convinced the efficacy of prolonged exercise to be due to the
change it produces in the blood, the "sanguis cibi," the
blood after a meal, being the most irritating to the lungs
of asthmatics; the blood after fasting or prolonged exercise,
having lost all its "recombinatius materials," being
in an opposite condition; least so 2. So do not think this
is the correct explanation; for after a hard day's exercise
the asthmatic requires generally takes a hearty meal,
without in any way vitiating the beneficial result.
Moreover some asthmatics are not affected by metabolic
conditions, at no amount of "sanguis cibi" is capable
of inducing an attack. I have known several instances,
I think the efficacy more probably lies in the thorough
fatigue and weariness involved, blunting the sensibility to
the slightest external stimuli which usually are sufficient
to excite a paroxysm, for the exercise must always
be to the extent of absolute fatigue.
true case should be taken to avoid colic, as a slight
bronchitis in many patients will always excite
asthmatic attacks, he some also damp feet; in other
the breathing of cold air will bring on asthma. In
the latter case breathing through a respirator when out of
doors in cold weather, or a pie in the bedroom at night,
may effectively keep off the attacks. Except in those
special cases however, a pie in the bedroom is not to be
recommended, as it greatly increases an already over-
sensibility.

Regularity, as to meals, habits, hours, is also of great
importance to the asthmatic, for in very many cases any
little irregularity in these matters is apt to bring on
an attack; and careful attention to diet, regulation of the
bowels, are all that are necessary in the way of
treatment. The digestion in asthmatics generally appears to
be capricious, irregular, the slightest error in diet or
a late dinner, or a snuff, are quite sufficient to cause
a paroxysm. During an attack the state of things is
quite exaggerated. The dyspnoea caused by taking food
is so severe and distressing, that patients have absolutely
starved themselves for 24 or 48 hours, till the attack passes
off. The great points are not to go to bed with food
lying indigestible in the stomach; to make the last meal
of the day a light one, consisting of plain, easily digestible
food; to allow at least 5 or 6 hours to elapse after
this before going to bed.

Dr. Hope Salter advises the following as the most suitable diet
for an asthmatic:

**Breakfast.** A small basin of breakfast-cup of sweet milk, a
berrie this an egg, (two for a strong man with a good appetite)

or a mutton chop, or some cold chicken or game. As a drink, if any is required besides the usual milk, tea is better than coffee, cocoa, than tea, and milk water last of all.

Dinner. Not before 2 or after 4. But mutton be the staple meat, beef or lamb but rarely, pork or veal never. A little succulent vegetable or potato should be taken; a little jammages, fruiting, or stewed fruit or the fruit at a fruit should conclude the dinner. Only one helping of either meat or fruiting. Water is the best to drink. No cheese or dessert. This should be the last meal; nothing of all should be taken after this till the next morning, or is absolutely necessary only a little tea with a cutter.

"Breakfast should be made the main meal of the day and should be taken early ag. 8 a.m.

Meat is especially bad after to bring on attacks and should never be taken as any late meal. Fish suits most patients, especially salmon. Stimulants of any kind are as a rule bad.

There are especially mingerous articles, grief, which frequently bring on attacks. Should be avoided by all asthmatics, e.g. all preserves, potter meat, fish, tongue, sausages, stuffing, seasoning, pepper, piquant condiments, orange peel, treacle, mints, almonds, raisins, cheese, meat preserves, steak batter, coffee.; heavy malt liquors, especially those containing alcohol, or carbonic acid gas such as bottled stout and Scotch ale, are the worst of all drinks for asthmatics."

Each individual case must be the subject of careful study, treated on its own merits, in many cases most of these restrictions would be unnecessary; many patients
patients can take a late meat dinner with perfect impunity, whereas others do not dare to touch meat after 2 p.m., or even food of any description.

We now pass on to the treatment of the Parotism.

**Treatment of the Parotism.**

Here we are beset with difficulties; there is no drug which we can rely upon to always be good for in one case a certain drug may bring absolute relief, from absolutely useless or even harmful in another. All we can do is try the various remedies till we find out the one which brings the most relief in each individual case. In some all appear to be of little or no avail, sometimes even when we have discovered a remedy which brings relief its efficiency gradually seems to wear off, until it too ceases to do any good.

It is sometimes useful in order to avoid this, to vary the treatment from time to time, “ringing the changes” on two or three drugs.

The first thing to do is to remove any possible exciting cause, e.g., relieve a burning stomach by an emetic, or a burning bowel by a saline aperient; exchange a feather pillow for a horsecandy one or a straw bolster, try the effect of removal to another bed or on to a sofa, or into another room if possible. It very often happens that the sufferer on moving from the bed to a sofa gradually gains relief. If he is able to lay off comfortably to sleep, see that the room is not close; if not, try the effect of lighting a fire.

Of all such simple experiments, few, all we can do is
to put the patient into the most comfortable position for respiration, e.g. in an arm-chair; or with his arms resting on a table—this often brings great relief; and enables him after a time to drop off to sleep with his head on a pillow on the table, till the attack passes off, & he is able to return to bed. If the hypnura prevents him from getting to warm, let him rest his hands on a table standing up, or he may even have to hold on by the mantelpiece, or a towel fastened to the lip of the bed-foot. Always see that as far as possible, he is clothed sufficiently to prevent his catching cold.

We must then try the hypnura remedies as our first choice, beginning with those which are most commonly successful.

Dr. Goodhart recommends Pot. Sodic. & Tinct. Belladonna.

To begin with, a or Eet. Sodic. 1/2, or 2x-xx every 1/4 hour, for two or three doses at the onset of the attack. This latter must be taken in milk or sweetened water, otherwise the resin separates & sticks to the vessel.

Professor Granville Stewart recommends 4y. Pot. Sodic. 3y, Ammon. Carb. 3y, Ag. at 3y. Sag. 3y every 2-3 hours or 8 & Pot. Sodic. gr. 3xxvi, Eet. Sodi. qu. 3xxvi, Eet. Glycer. qu. 3xxvii, Chlor. Ames. 3y, Ag. at 3y. Sag. 3x every 4 hours.

Perhaps the best way is to begin with Pulv. Speear. gr. 3x, as this is often successful, then pass on to the inhalation of some of the various gases, powders, etc. which is often most useful. These always contain nitric acid, especially stramonium also. The simplest are "inhalation papers," which are made by saturating white blotting-paper of mercuric thickness with a solution of Pot. Mercur. gr. 30-40=60-60 gr. to 3y of water, having iv.
Dr. Hyde Saller recommends Dr. Nitrate Zirr at Aq. Oph. In "Ozone Paper", which are composed of a mixture of chloride nitrate of potassium made to look like "artificial pearls", which are of similar composition. In "Humphreys" or "Hickings", or "Bliss", or the "Great Mountain" Cures, these may all be accurately imitated by "Podo. Hotel. Comp." The composition of which is as follows — Dr. Nitrate, or Aq. Decid. Bull. ox. Zirr. Assorted to a bath in fifteen. Stramon leaves in powder, black tea in powder — ox. Zirr. Mix well, try, and take it. Amis. H. IV. Suy. The fumes of Zirr a smoke, burn on a plate to be inhaled 6 or 8 times a day, the bedroom fumigated with the same.

"Humphreys Cure" is composed as follows —


Another useful preparation is the following —


The fumes of these various preparations very often from extremely beneficial and should always been given a trial. It is important to use them early when the first symptoms of the paroxysm appear, for they may succeed in the early stages though they fail later. The room should be small. Their efficacy is due to the generation of a nitrate by
by combustion from a nitrate; the nitrate paralysing the muscular fibres of the bronchial tubes. They usually act quickly, exciting some cough at first, the breathing becoming easier in a few minutes. Sometimes they give only partial relief, sometimes they succeed at first but fail later, often they fail altogether.

In some cases the inhalation of the fumes for a few minutes before going to bed, proves an effectual preventive.

The smoking of the Batna stramonium for asthma, was first introduced by General Court from India in 1802. It is usually its efficacy to the generation of a minute from a nitrate by combustion. In some cases it is far better than the remedy, in a great many it acts benevolently to some extent, but in a few it is harmful. Very frequently gives only temporary relief. Hence other remedies it is important to administer is early, as it will cut short an already arisen spasm, when it will fail to affect a paroxysm of any duration.

It may be smoked in a pipe, or in the form of cigarettes. In some cases, when merely smoking it has failed, the inhalation of the cold smoke - i.e. fumes from a pipe with a tumbler and inhaler - has proved successful. If the Batna stramonium fail, the more powerful Batna jurt may be successful, or the Batna talata, which possesses still more active properties.

Sometimes the leaves fail altogether, but the seeds, which possess much more of the active principle than the fumes, are efficient in using the seeds, however great care is necessary, they must be smoked in very small quantities, gradually increased, the effects carefully observed. (Hogg & Wall.)

Stramonium may also be given internally in the form of
of the extract beginning with doses of gr. ½, or the tincture beginning with M½ doses. As with the previous remedies it should be remembered that in some cases it may prove a valuable preventive, e.g. by smoking a pipe after the last thing at night, whether any arthritic symptoms are present or not.

Nitrate of Somm, internally, in doses of 2-3 gr. has a similar action.

Nitroglycerine – a triminate of glycerol – acts more powerfully than other nitrites – its efficacy being due to the production of a narcotic minute, probably because the whole jet is absorbed without decomposition, the nitrous acid is thus set free in the blood itself. It should be administered in doses of gr. too.

Amyl Nitrite has an action similar to that of other nitrites, but it is very transient in its effects. It acts very rapidly, bringing complete relief in a minute or two, but unfortunately its effects very soon pass off and the symptoms return as fast as ever. It should be inhaled in doses of M½–V.

Ammonia bichlorate, is a substance which proves very successful in many cases. It must be given in large doses of 3½–11½ up to 31½ of the tincture, every 2–3 hours till the effect is produced. Small doses cause contraction of non-striated muscle, but large doses paralyse it. Slightly 8:05 pulmonary and heart are also paralysed. Hence it must be used with caution in heart cases. It produces a very similar effect to that of vis ally, tobacco, viz. faintness, giddiness, nausea, cold sweat, great depression, it must be used till these symptoms appear – hence it is a 1. Parry Brumton. "System of Therapeutics."
It is a somewhat severe remedy. In non-smokers, the depression produced by smoking tobacco or marijuana, is extremely efficacious in relieving the spasm. It is a severe remedy, care must be taken not to exceed it, a very serious nostrum collapse may be induced; it is quite sufficient to produce dizziness, faintness, cold sweat, nausea; when the spasm will at once cease. The best way is to smoke some mild tobacco in a pipe, for want, held in a cigarette or two should be sufficient. An ethereal mixture of tobacco may also be given internally.

Though a severe remedy, tobacco may prove a great boon in some cases of violent intrauterine spasm. In hay-fever, asthma, it is sometimes invaluable. Unfortunately, the patient soon becomes habituated to its use.

The inhalation of pyrethrum - a volatile bony contained in tobacco smoke has also been highly recommended. It is placed on a plate in a small room in which the patient sits for 20-30 minutes, thrice daily. In some cases, all symptoms disappear entirely after a few sittings.

One of the simplest and in some cases, most efficacious of remedies is hot strong coffee. Its effect varies greatly; often it brings only temporary relief, which is most slight; but sometimes, complete and permanent so far as the single attack is concerned. It must be given very hot, in very strong, without sugar or milk, not in too large a quantity, on an empty stomach; for if on top of a meal it merely aggravates the condition. Valerian or caffeine in doses of 0.5-0.75 has also proved of great value.

Another remedy which in certain cases is invaluable is alcohol. It must be given in a considerate dose, but unfortunately has to be gradually increased to maintain its efficacy. There are however some distressing cases of violent asthma, in which it gives very great relief when everything else fails, in which it is justifiable to prescribe it. It must be given hot strongly on an empty stomach.

Chloroform again is an agent of great value in some severe paroxysms, when everything else has failed. The sufferer appears to be in extremis. It should be given gradually and cautiously, never to produce insensibility. Great care must be observed that the patient is not allowed to administer it to himself, for he may in the distress of a paroxysm from our a large quantity, and gradually go into the inhalation, fall asleep, and with a fatal result, as occurred in a case mentioned by Dr. Tooke. It has several disadvantages; for it gains a hold over the patient like opium, and the dose requires to be gradually increased. It often induces very troublesome secondary results, also quite prolonged nausea, incessant respiration, troublesome secretion of saliva, tinnitus, apathy, generation. Moreover, if taken in a prolonged period in large doses it appears to increase the arterial tension, even to produce a dyspnea of its own, either its discontinuance is necessitated. It has however proved useful in small doses as a preventive, as in a case related by Dr. Russell Reynolds of "a young lady who by inhaling a few drops on her handkerchief whenever I, "Medical Gazette," Dec. 1850.
whence an attack threatens, at once act or it; in the extreme cases mentioned above it may justifiably be resorted to.

Ether may also be used in a similar manner with the same restrictions and precautions.

Opium is a remedy which has been very highly praised, occasionally in extremely severe cases it is very indispensable and justifiable. It also has many disadvantages; it causes nausea, disturbs the digestion, soon becomes insipid, so that the dose has to be increased. Some other evils may be counteracted by combining it with atropine gr. 1/20 or gr. 1/720. When other remedies have failed the dyspepsia is extreme, it may be justifiable to use it. It is found in many cases to abort the attack; though sometimes it aggravates the symptoms. Certain patients may learn to use it only in the worst attacks, so maintain the efficiency of a small dose; Stevenson, e.g., in 5 years never increased the initial dose of gr. 1/6, which acted with uniform success. It is best given in the form of morphia subcutaneously in doses of gr. 1/4.

Similarly also with chloral, which has frequently been used with great success; it is admirable in certain severe cases when simpler remedies have failed. It must be given in large doses of gr. XV-XL; largely diluted, it acts quickly, in some cases may be indispensable. Great caution is necessary, in other cases it must not be used.

Cannabis indica has also proved invaluable at times, and in certain cases may be prescribed under similar restrictions.

Pellagra is a disease which has proved extremely beneficial in many cases. It is strongly recommended by Denbrock, Harby, Hyde Salter, Trousson and many others. It is best given in doses of one grain to the ounce, to begin with, gradually increasing or to tolerance. It should be taken at bedtime to anticipate the onset of the attack in the early morning. It may also be given in the form of atriplex, subcutaneously, in doses of quarto. Or it may be smoked in cigarettes with Stramonium.

The fumes of burning sulphur sometimes bring relief, though often they aggrandise the dyspnoea. Similarly also with the inhalation of ammonia, oxygen, Compressed Air, of Carbonic Acid Gas, all of which have proved useful, though generally they of no value.

Many other remedies have from time to time been recommended as "acting like a charm," but these are the most trustworthy, the most likely to prove beneficial. The best we can do is to begin with the most commonly successful, find out which gives the most relief in each individual case.

In cases which are complicated by bronchitis, emphysema, or cardiac disease, our difficulties are greatly increased, as we have these to treat also. In bronchitic asthma, every effort should be made to curb the bronchitic discharge of air, without losing sight of the asthmatic element.

"Grugal asthma" can almost always be cured by removing the patient from the influence of the exciting cause—e.g., a sea-voyage in the case of hay—if this is impossible we may palliate the discomfort by various means. 1

Snuff, inhalations &c.

In old confirmed, advanced cases with various pulmonary or cardiac complications, putting beyond a certain amount of palliation of the distress is possible. For these specially constructed arm-chairs to rest and sleep in offer great comfort.

The treatment of the asthmatic paroxysm is thus seen to be very unsatisfactory, it must necessarily be so; for it merely alludes the same relation to asthma, that the epileptic fit alludes to epilepsy. The only rational treatment must be to attack the essential cause of the disease, i.e., the asthmatic tendency.