SOME THOUGHTS AND EXPERIMENTS ON RESPIRATION AND ON ASTHMA, WITH SPECIAL REFERENCE TO HENRY HYDE SALTER

by

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> Nothing can be more terrible than a paroxysm of Asthma: the organs of respiration, which are the pillars of life, are shaken to their foundation and the entire destruction of the machine is apparently threatened.
>
> THOMAS WILLIS (1621–75).

PLATO said that the 'Genii placed the lungs in the neighbourhood of the heart, to keep it cool and in exact obedience'. Galen was near a truth—'the thorax, distending, draws in the air, the lungs following the dilatations of the chest'. One of Harvey's teachers at Padua, Fabricius (1537–1619), at the beginning of the seventeenth century, explained correctly the action and properties of the diaphragm. In the two centuries following the discovery of the circulation of the blood speculation and uncertainty concerning the action and movements of the lungs continued. Robert Boyle (1627–91) was impressed with 'the diaphragm which forms the principal instrument of ordinary and gentle respiration although aided by the intercostal muscles and perhaps some others'.

Fig. 1. 'When the vital capacity of the lungs is to be made, let the person to be examined loose his vest, *stand perfectly erect*, with the head thrown well back; then *slowly* and *effectually* fill his chest with air, or *inspire* as deeply *as possible*, and put the mouthpiece between the lips (standing in the same erect position), holding it there sufficiently tight as not to allow any breath to escape; the observer in the mean time turns open the tap: immediately the patient empties his lungs, and *slowly makes the deepest expiration*; at the *termination* of which the operator turns off the tap, confining in the receiver the expired air, which part of the Spirometer is now raised out of the reservoir.' JOHN HUTCHINSON, 1846

Fig. 3 'Let that portion marked H represent the "residual air", or air left in the lungs after a complete voluntary expiration; the part next anteriorly marked *white*, the "reserve air", or space for all that air left in the lungs at the termination of an ordinary expiration. The black stripe, the space for the ordinary "breathing air"; the white next anteriorly, the portion for the "complemental air", or extreme deep inspiratory movement; and these three, viz. the complemental, breathing, and reserve airs, conjointly, we style, for convenience, the "vital capacity", in contradistinction to "absolute capacity", which may be considered as the whole *four* divisions combined.

'*The residual air* is entirely independent of the will, and always present in the chest.

'*The reserve air*, to use a simile, is a "tenant at will".

'*The breathing air* is constantly passing out and in, many times in a minute.

'*The complemental air* is seldom in the chest, and, when present, it is only so for a brief period. Nevertheless, the air commanded by these movements is constantly interchanging or transfusing.' JOHN HUTCHINSON, 1846.

In 1667 Richard Lower correctly described the respiratory act—'making a dog breathe like a broken-winded horse, by dividing the phrenic nerves'. John Templer (1672) observed and commented upon the 'multitude of the ramifications of the bronchial and sanguineous vessels' in the lungs. Borelli (1679) was the first physiologist to establish an experimental inquiry into the quantity of air 'received by a single inspiration', and James Keill (1708) made some near-correct cubic measurements of the air expired. In the years after 1757 Black, Rutherford, Lavoisier, Priestley and Scheele threw much light on respiration by their investigations into the composition of atmospheric air and respired air.

Thomas Addison and Thomas Hodgkin, physicians to Guy's Hospital (*c.* 1840), made observations on the anatomy of the lungs (with added impetus after Laennec's introduction of the stethoscope) and noted that a lung lobule is connected with the bronchial system by a 'minute filiform tube which abruptly terminates'—an excellent concept of the terminal bronchiole and the alveolar duct.

The next step was made by John Hutchinson of Newcastle (1846) by experiment on the capacity of the lungs and the respiratory functions by the use of a spirometer: this work is intensely interesting and original. He distinguished the divisions of the thoracic movements and analysed the 'vital capacity'; clarified the combined movements and the co-ordinated thoracico-abdominal action in respiration; observed and measured the range of respiratory movement in different depths of the respiratory cycle, and by testing a large number of persons, including 'firemen, wrestlers, gentlemen and dwarfs' (1844), concluded that 'capacity of the lungs increases in an arithmetical proportion to the *Height*'. This observation was soon followed by another, based on his appreciation of the value of determining accurately the volume of air breathed out at *one full expiration*, together with the power exercised by the muscles of expiration and inspiration concerned in the effort. Moreover, differentials were measured in respiratory action with the back fixed and the back free—he showed that freedom for whole body movement greatly facilitates overall respiratory excursion. In 1860 T. Graham Balfour followed up these studies and carried out an extensive investigation of the Grenadier Guards with spirometry. It is noteworthy that the two series of measurements of the mean vital capacity were remarkably close. Balfour agreed with Hutchinson 'that the spirometer might be advantageously employed in judging the fitness of recruits for the army'. About eighty years later, anatomists (Macklin, Pratje, Huizinga, Hasselwander, Brückner and Hayek) were able to prove the nature and extent of the movements of the mediastinum, chest wall, trachea, bronchi and diaphragm: in respiratory movements (with the whole body quite free) all these structures move according to the phase of respiration and in degree according to the age of the child, for the angles of the ribs and the freedom for descent of the diaphragm vary with age.

Although Laennec (1821) rather underrated inspection of the movement of the chest as a means of diagnosis, he predicted that

> the extended use of the stethoscope would enable us to trace many cases of asthma, now considered to be nervous, to particular reasons now little known . . . where breathing is habitually

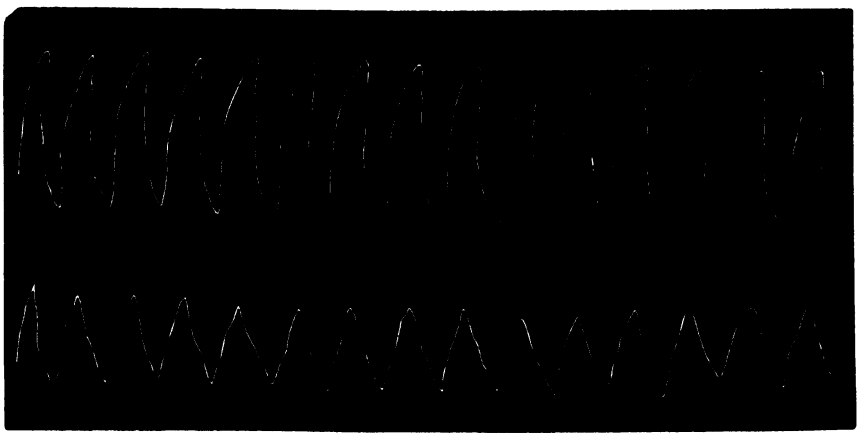
Height.	Guards.		Hutchinson.		
	Number of observations.	Mean vital capacity.	Number of observations.	Mean vital capacity.	Capacity calculated in arithmetical progression.
5 feet 7 to 5 feet 8...	17	225·53	411	228	230
5 „ 8 to 5 „ 9...	315	237·51	329	237	238
5 „ 9 to 5 „ 10...	400	242·04	201	246	246
5 „ 10 to 5 „ 11...	235	249·16	116	247	254
5 „ 11 to 6 feet.....	113	253·75	112	259	262
6 feet and upwards...	46	264·00	80	276	270

Fig. 4
Studies of the Vital Capacity in relation to height.
J. Hutchinson's series were taken in 1846. The Grenadier Guards were tested by T. G. Balfour in 1850

Fig. 5
Simultaneous tracings of the movements of the fifth and second ribs in an adult male, made by the stetho-cardiograph

A. Ransome, 1873. 'The movements of the second rib are less extensive than those of the fifth, this being characteristic of the "inferior costal" type of breathing in the male'

Fig. 6
Tracings of the movements of the second and sixth ribs in an adult female, forced respiration



'In females, displaying as they do the "superior costal" type of breathing: and the female respiratory curves are more abrupt in their rise and fall.' A. Ransome, 1873

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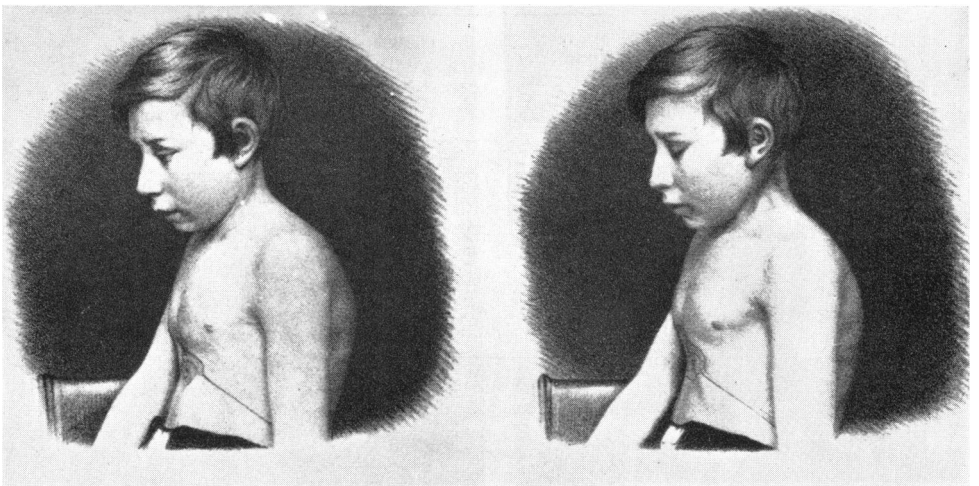

Fig. 7
Boy aged 13 years. Chest deformity arising from persistent 'inspiratory obstruction'—'the walls of the chest recede during inspiration' and the 'inspiratory muscular efforts are much exaggerated'
Francis Sibson, 1848

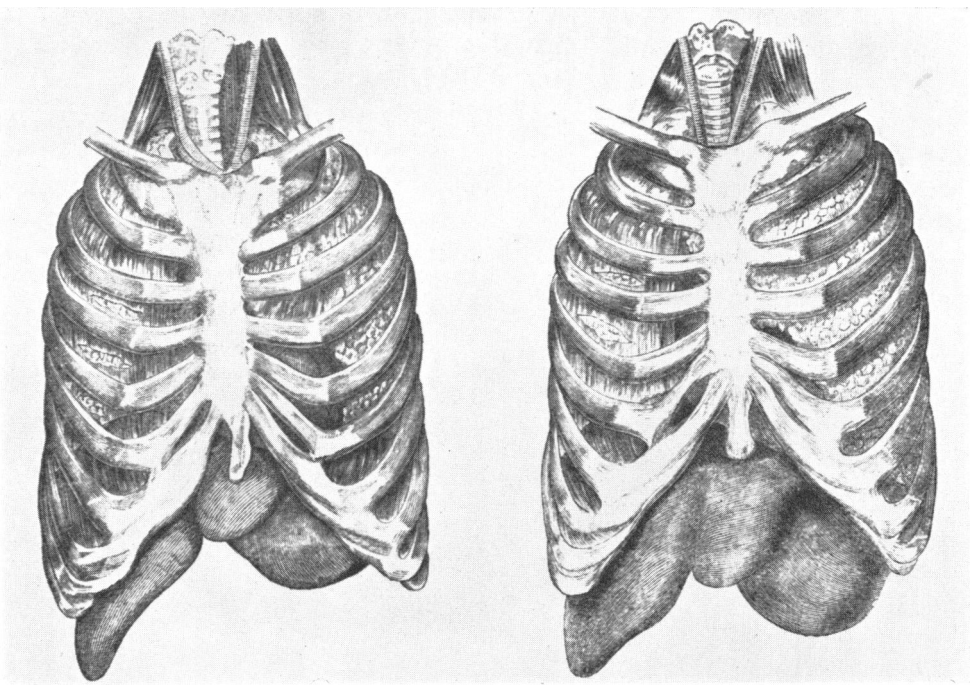

Fig. 8
The excursion of the thorax in inspiration. Francis Sibson, 1848

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difficult and oppressed . . . under the same head of 'purely nervous' may perhaps be ranged the 'spasmodic asthma'—an evening paroxysm, and a moving remission, after a slight expectoration.

Moreover, Laennec's *Mediate Auscultation* enabled him to note that the breath sounds were of a special kind in those 'which cannot be said to have any actual disease of the lungs but soon get out of breath and are very liable to catch cold and to have sibilant rattle—or having the constitution to which the name "Asthma" has been given'. Francis Sibson (1848) closely observed the thoracic respiratory excursions and especially where obstructive phenomena were present. The anatomical complexes of mediastinal and hepatic descent were accurately recorded. The synchronous ascent of the ribs was measured by A. Ransome. William Jenner (1857), following up the attentions which physicians had fixed on vesicular pulmonary emphysema, and recognizing that 'vesicular emphysema as being incapable of cure, its prevention is of the highest importance', formulated the principle that vesicular emphysema is the 'invariable consequence of whatever impedes the free exit of air from the lungs and at the same time excites powerful expiratory efforts'. Of this, however, Laennec had been well aware—

the disease (emphysema) consists in an excessive, permanent and unnatural distension of the air cells . . . but when the distension becomes still more considerable or takes place with greater rapidity, the air cells are ruptured at certain points . . . and, the bronchial tubes, especially those of a small calibre, are sometimes very evidently dilated in those portions of the lungs where the emphysema exists . . . as detected by the best practical morbid anatomists.

We are indebted to H. H. Salter (1860) for many original studies on asthma. After defining the five conditions of normal respiration—a certain length of respiratory interval, a certain range of movement, a certain ratio of parts of the act, certain moving powers, and a certain sensation—and stating his ideas on 'the forces concerned with respiration', he measured intratracheal pressures during respiration and obtained experimental support for his 'Co-efficients of Respiratory Statics' (Table 1).

TABLE 1

CO-EFFICIENTS OF RESPIRATORY STATICS. (H. H. SALTER, 1865).

At level of	*Forces tending to Expiration*		*Forces tending to Inspiration*
Extreme Inspiration	Elasticity of lung Physical reaction of parietes Gravitation of chest wall	extreme	Muscles of extraordinary inspiration.
Ordinary Inspiration	Elasticity of lung Elasticity of parietes Gravity of chest wall	equilibrium	Muscles of ordinary inspiration.
Ordinary Expiration	Elasticity of lung Gravity of chest wall		Elasticity of costal parietes.
Extreme Expiration	Elasticity of lung Gravity of chest wall Muscles of extraordinary expiration		Elasticity of costal parietes. Extreme.

R. Bree (1797) contended that in asthma the obstruction is a temporary one and the symptoms disappear with the irritation: the bronchi, whose internal

membrane is intended to 'effuse a light lubricating moisture only', may fill with 'lymph' and the passage of the air through these narrow pipes becomes impaired —'hence wheezing', both in inspiration and in expiration. Salter disputed Bree's suggestion that the asthmatic paroxysm was merely an extraordinary effort to get rid of some 'peccant and irritating' matter existing in the air tubes—a sort of bronchial tenesmus—Salter reversing the idea, thus—

the stethoscope would have shown him [Dr. Bree] that the conditions of an extraordinary discharging power are not present in an asthmatic attack; indeed, the power of getting rid of anything in the lungs is very much diminished by asthma, and, if any one who listens to the chest of an asthmatic during a fit of his disease will note that the amount of air respired is very small indeed, much below the natural standard, and that the vesicular breath sound, even when not drowned by 'rhonchus and sibilus', is either inaudible or very feeble, and he will see that the amount of movement of the parietes of the chest, in spite of the violent muscular movements and its great distension, is very slight. Moreover, if the patient tries to blow his nose or to cough he finds he cannot make a sufficiently full inspiration or get enough air into his chest to perform the act efficiently—he can make only little, short and almost soundless blows which achieve nearly nothing.

This is an almost precise statement of our modern concept of 'air trapping' occurring in forced expiration and coughing in asthma. But, Salter goes on to even more clarity of description when he says:

the inordinate mucous secretion, the expulsion of which gives so much relief at the termination of a paroxysm of asthma, is, in part, a result of the congestion into which the lung capillaries have been thrown by the long-continued imperfect respirations, and the secretions, . . . and the relief of these congested capillaries and the release of accumulated mucus, when the bronchial spasm ceases, enables relief of dyspnoea . . . expectoration is, therefore, attended with relief . . . this expectoration never takes place without a marked abatement of the dyspnoea, for the simple reason that until dyspnoea does abate, and until bronchial spasm is passing off sufficiently to allow the chest to be freely filled with air, efficient cough, adequate to the free discharge of this accumulated mucus, cannot be effected . . . expectoration cannot take place till the intensity of the dyspnoea is already subsiding. [Salter sums up the order of events] air shut off from the lungs by bronchial stricture; consequent pulmonary congestion and a state of partial asphyxia; abundant mucous exudation; inability to expectorate this material because of inefficient cough; abatement of bronchial spasm; recovery of cough power; free expectoration; complete relief.

M. Beau (1850) had regarded the wheezing and the dyspnoea to be dependent on bronchial obstruction by inflammatory products. Much argument ensued for many years whether the secretion, exudation or a bronchial spasm, was the primary cause. The stethoscope was used to settle the question, W. T. Gairdner (*c.* 1850) putting it: 'There is no asthma without bronchial spasm which causes sibilant rhonchi in expiration and in inspiration, thus proving the obstacle to the entrance and especially to the exit of air to the lung vesicles', and, with the evacuation of 'a thick semitransparent mucus and cessation of the asthma, these auscultatory signs disappear'.

Yet, even a nervous element, 'nervous asthma', was considered important by Laennec. The whole truth was being gradually discerned. W. Cullen (1712–90) had suggested the idea of 'spasm' chiefly because he was unable to explain the symptoms of asthma in any other way. G. Budd (1840) was open-minded:

There still, however, remain some cases, which at present we can only explain by supposing the dyspnoea to be nervous . . . it seems probable that the number of such cases will be still

further diminished and that many of those 'fits of asthma', which we now generally consider nervous, will be discovered to depend on some change which has yet escaped our observation . . . if the 'fits of asthma' are really nervous the difficulty must result from bronchial spasm and from some suspension of the normal action of the diaphragm and other muscles of inspiration.

Here was clinical insight. Many had concluded, on the evidence, that the difficulty of getting air out of the chest in asthma is much greater than that of getting it in . . . and Budd was moved to say:

that however rapid we make our breathing (voluntarily) we perceive no difference in the ease with which successive acts of inspiration are performed . . . this circumstance would seem decisive that the fibres of the bronchi have, normally, no independent rhythmical motions of contraction.

—a meaning which I interpret to be that there is no inco-ordinate overtone or hypertony in the normal bronchi-bronchiolar system. It was suspected that there is slight contraction of the whole bronchial system at each expiration: in fact, Salter postulated a slight dilatation of the air passages in inspiration and slight contraction of the air passages in expiration, but this would 'normally produce no abnormal sounds', and all this 'looked as if inspiration opposed and expiration favoured contraction of the air passages', but in health this was simply due to a simple recoil due to the tendency of their muscular and elastic walls to contract—that bronchial contraction and inspiration, normally, could not coexist. Salter and some of his colleagues, particularly W. T. Gairdner, revealed remarkable awareness of other aspects of bronchopulmonary action.

In relation to cough, the opinion has been advanced that the muscularity of the bronchial tubes may, by diminishing their calibre, increase the rapidity of the rush of air driven through them by the act of coughing, and thus increase its expulsive power. If this contraction were general and extended to the smaller tubes, the reverse would be the case, for a smaller stream of air would be brought to bear upon the obstructing material. If, however, it is a circumscribed contraction, confined to the situation of the matter to be expelled, then it would be a veritable adjuvant, and the air would rush through the point of narrowing with increased rapidity, and therefore increased expulsive power, just as narrowing an outlet of water clears and deepens the channel. But the contraction being at the seat of the matter to be expelled, and there alone, is an essential condition to this increase of expulsive power. A little glottis is, as it were, formed there, and the material inevitably driven through it.

Salter also considered that the 'ultimate bronchiae', i.e. smaller bronchial passages, must have a different mechanism for expulsion of mucus and accumulated secretions than that which occurs in the larger air-passages which are emptied by the velocity of the air flowing through them. He considered the ultimate bronchiae are emptied by peristaltic contraction. Salter's concept of a dual mechanism for emptying the bronchial passages is now remarkably well supported by ciné-radiographic observations of the removal of oily 'dionosil' contrast medium by expectoration in patients, following bronchography and performed a hundred years after his original remarks. Thus, in 1957, Holden and Ardran noted that 'coughing will only clear contrast medium from the trachea, main and medium sized bronchi and this can be effected in a short time. Clearing of the small bronchi and bronchioles takes place at a much slower rate.'

Salter had written

We see that the purpose of this muscular furniture of the bronchial tubes is that they should contract under certain circumstances, and on the application of certain stimuli; and seen by this light we recognize in asthma merely a morbid activity—an excess—of this natural endowment; the tubes fall into a state of contraction with a proneness, a readiness, that is morbid; the slightest thing will throw them into a state of spasm, the irritability of the muscle is exalted, the contraction violent and protracted, that becomes a stimulus on contraction which should not be, and the nervous and muscular system of the lungs is brought within the range of sources of irritation applied to such distant parts as ordinarily in no way affect them. A greater degree of bronchial sensibility is shown in those cases, by no means uncommon, of what is called 'hay-asthma', in which the stimulus to bronchial spasm is the effluvium of hay; a still greater is that numerous class of asthma in which the disease is called into activity by certain atmospheric peculiarities which are altogether inappreciable, as where an attack of asthma is inevitably brought on by going to a certain place, living in a certain house, sleeping in a certain room. All these cases fall strictly under what we may call the formula of health; they are physiological; they are instances of the contraction of a muscular tube in obedience to stimulus applied to the mucous membrane that lines that tube; the nervous system engaged is the *intrinsic* nervous system of the tubes, its own ganglia and perceptive and motor filaments; the error is merely a morbid exaltation of a normal irritability. But there are other cases in which the error is more than this, in which the nervous apparatus involved in the phenomena is abnormally extended; in which certain outlying and distant parts of the nervous system are the recipients of the stimuli that give rise to the bronchial spasm. In the former class of cases the bronchial spasm takes place in obedience to the wrong stimulus applied to the right place; in the latter, place and stimulus are alike wrong; the relation of the asthma to its cause is in the one case immediate or primary, in the other, remote or secondary,—mediate, through the intervention of some part of the nervous system extrinsic to the lungs. At any rate, it is clear that the vice of asthma consists in the irritability of the part irritated. (Salter, 1860.)

The genesis of the spasm theory led Salter and others to select such remedies 'as appeal to the nervous system', e.g. 'antispasmodic sedatives and direct nervous depressants', perhaps (historically) the most striking being 'chloroform —a few whiffs, and the asthma is gone: a dyspnoea that a few seconds before seemed to threaten life being replaced by a breathing calm and tranquil!' And, the effect of an emotion—'a shock may bring on an attack—or a shock may cut short an asthmatic spasm . . . the sight of a dentist's house may be enough to cure it'. Arguments flowed in favour of the idea that 'the periodicity of asthma implies its nervous nature'; and 'the possible absence of appreciable organic change by post-mortem examination, even where the disease was of some duration, seemed in favour of an essential nervous factor'. It was asked,

what if, at *post-mortem*, there be no trace of inflammation or its products, the vesicular structure quite healthy, the passages leading to it lined by a healthy mucous membrane, the cavities of the pleura free from all abnormal contents, and the heart sound—if the disease in fact shows no cause, where then shall we locate it? What is the starting point?; if there is no organic trace may we lay it down to the nervous system?

In 'pure emphysema' it is noted, the dyspnoea remains, varies but little, and has no wheeze. But the dyspnoea of asthma 'tells a plain tale', the wheezing giving positive evidence of narrowing of the air-passages: the breathing being anciently described as 'tight'. Salter, with his insight, understood:

hollow tubes give no musical sound, when air rushes through them, if they are of even calibre, but if they are narrowed *at certain points*, if their calibre is varied, the air in them is thrown into

vibrations and they become musical instruments—the wheezing of asthma, then, is a positive evidence of a bronchial contraction, it is a physical demonstration . . . this may and could be *by contraction of the circularly-disposed muscle* which exists in the bronchial walls, and this would explain the 'sibilus' of asthma.

It could be of the nature of an 'excito-motory or reflex action', as in a case reported by D. Chowne in 1850, in which 'the application of cold to the instep immediately produced the asthmatic condition'. Conditioned states were being recognized. 'Psychosomatic' phenomena were being appreciated but, of course, the *modus operandi* was occult.

Of the significance of the sputum in asthma, Salter records (1860):

If the attack is very short—an hour or so—the expectoration of a pellet or two of mucous is all that takes place; if it lasts the entire day, in addition to the expectoration in the evening which winds up the attack, it will appear again the next morning and even for two or three mornings after; if, however, the attack is protracted over several days, the expectoration may continue for a week or two. It is curious after how slight and transient a tightening of breathing, this mucous exudation will invariably appear. Many asthmatics are liable, after laughing, or after taking food, or inhaling dust or smoke, to little paroxysms of the true asthmatic dyspnoea, lasting perhaps five or ten minutes, and then over; each of these little attacks is sure to be followed in a few minutes by the expectoration of one or two little portions of this mucus, sometimes so immediately that its discharge appears to be the occasion of the relief of the breathing. In character this mucus is very curious; it is in distinct little pellets that are coughed up and expectorated with the greatest facility, unattached to anything else: they are of the consistency of jelly and thick arrow-root, of a pale grey colour, of an opalescent transparency, and a saltish taste.

On examining a portion of this material with the microscope it is seen to consist of a nearly homogeneous viscid matter, and in looking at some specimens you might almost imagine you are looking at pus. Are they pus cells?

In this there was included a suspicion of another factor—but bacteriology was yet to be discovered.

Salter inquired of the periodicity of asthma—was there an underlying 'diathesis'? Asthma in winter meant muscular spasm engrafted on inflammation in the air-passages. But sometimes asthma was worse in hot weather. The periodicity was of two kinds, '*intrinsic* and *extrinsic*, the latter dependent on the periodic recurrence of the exciting cause; the former, the true essential periodicity, independent of all external circumstances'—so much so that many asthmatics become thoroughly acquainted with his or her own case, recognizing the slightest indications and able to predict each event of the disorder, knowing perhaps when 'it is coming on' and when 'it will go off'. But it may be that 'symptoms that were occasional become more confirmed or more capricious', and in childhood 'asthma appears on a constitution possessing a congenital proclivity to the disease'. Even early infancy is 'accessible to asthma' and many of the 'purest spasmodic cases start from this early date', the constitutional tendency being strongly marked indeed that would develop itself so early, even suggestive of possible factors of inheritance in constitutional character. Nearly one in five may have signs in the first year of life. The aetiology was admitted to be 'most obscure and difficult' to comprehend. Salter held that asthma 'had

at the root of it some organic disease in the chest *or* that genuine spasmodic asthma depended upon no lung disease but upon a pure neurosis'. But, whatever the aetiology, there may ultimately be hypertrophy of the bronchial muscle, a thickening of the circular muscle, and possibly some permanent diminution in bronchial calibre, sometimes a degree of contraction 'much beyond what is explicable', which might be 'due to *recurrent or persisting bronchitis*', converting some of the tubes into 'fibro-muscular cords, thickened and knotty'; or, perhaps, a *dilatation of bronchial tubes*, the vital and physical properties of the bronchial walls having been *impaired by inflammation*, indicating that 'asthma and bronchitis' have coexisted. In such children the chest has become deformed as a result of long-standing impediment to free flow of air and the voice is feeble, sentences are short and frequently interrupted by a single dry cough.

Does the occurrence of asthma once render the subject of it more liable to a recurrence of it? It might be that a person who has had asthma once must have a predisposition to it and, therefore, it will be likely to occur again. An isolated single attack in anyone is rare. Is it a case of a 'mere viscious habit or is it that once the bronchial muscle becomes hypertrophied it becomes increasingly prone to take on a state of contraction?'

On the 'therapeutical influence of locality' Salter's many cases are illuminating, and he gave newer hypotheses concerning the fundamental quality inherent in the bronchial (bronchiolar) state which is the initial predisposing factor, namely an inborn basic bronchiolar hypertonic state, existing usually in a latent form but rendered manifest by an easy susceptibility to contraction spasm; an excess lability, in fact. This concept might be considered together with some of Salter's own 'brief conclusions' concerning the 'asthmatic habit'.

That residence in one locality will cure, radically and permanently cure, asthma resisting all treatment in another locality.
That there is no end of the apparent caprice of asthma in this respect, the most varying and opposite airs unaccountably curing.
That some of these differences determining the presence or cure of asthma appear to be of the slightest possible kind, arbitrary and inscrutable.
That, consequently, many healthy persons, who never have had asthma and never may, probably would be asthmatics if their life had been cast in other localities.
That the disposition is not eradicated, merely suspended, and immediately shows itself on a recurrence to the original injurious air.
That, from the caprice of asthma, the constancy of the results in any given case is often deranged.

We would all agree with Salter in the following:

In a disease whose tendency is so little generally understood as asthma, and which is so alarming and distressing in its manifestations, the ultimate issue of the case is often a subject of the most painful anxiety to the sufferer and his friends; it is therefore a matter of some importance that the physician should be able to detect the tendency of the disease in any given case, and to form something like a definite prognosis.

Until recent times, the prognosis was necessarily based only on the actual condition of the person, child or adult, and partly on the history, duration and

frequency of attacks, completeness or incompleteness of the recovery between them, and other clinical data.

It has been long understood that unfavourable influence must be inferred from a 'persistence of expectoration' in asthmatic persons. This used to be called 'Humid Asthma'. Chronic expectoration is a menace, 'Wherever there is mucous exudation (probably now regarded more accurately as, at least sometimes, mucopus), there is cough, and chronic cough is a bad sign in asthma. On the other hand, the lack of expectoration in a patient with a chronic cough may be a sign of failure of the cough mechanism.' The ability to expectorate, even mucopus, may be something for which to be thankful! It is very important to detect the direction which the disease appears to be taking. Is it becoming more, or less, intense? Or, as Salter put it,

> the loss and the acquisition of the asthmatic tendency is generally a gradual process, and the future of a case often but a reflection of its past history. If a patient can tell you that his attacks have mitigated in severity and are getting less frequent, you have, especially if he is young, one of the most hopeful auguries of his ultimate recovery. If, on the other hand, the disease is *gaining* on him, you have what must be considered a very bad sign, and one which, unless some speedy and great change is induced by some of those means which control asthma, will leave but very little hope. If the lungs are entirely free from any organic complication, if there is no emphysema and no tendency to bronchitis, the patient has, under proper management, a very fair chance of recovery.
>
> Now, why is this? Why should age have such a determining influence on the tendency of asthma? In asthma, as in all other constitutional disorders, we have in the young much more room for hope from those changes in the type and build of the constitution which in them are so marked and striking. Indeed the existence of a constitutional peculiarity in a child is of itself almost a presumption that he will one day lose it.
>
> But there is a special reason, depending on the nervous nature of asthma, that makes us sanguine of recovery in the case of the young. What, for want of a better name, we must call 'nervous irritability', is much more marked in the young. Sources of irritation in the young are often adequate to the production of the most violent nervous phenomena. Thus the diminution of nervous irritability, as childhood passes into youth and manhood, may make an attack of asthma less and less prone to occur and less intensely spasmodic when it does occur. I believe, indeed, that this diminution of nervous irritability is the true explanation of that gradual recovery of young asthmatics which is so common, so almost universal.
>
> The prognosis is related to our ability to *detect the exciting cause* of the attacks, and the controllability of that exciting cause. (Salter, 1860.)

We now know, of course, that 'bronchitis (and bronchiolitis) with asthmatic breathing' in childhood demands the greatest diagnostic and therapeutic perspective, and the study of the child's abilities in respiratory exchange and in effective cough need special investigation. For the part played by a cold in the development of 'latent' state to manifest asthma, I refer any interested reader to Salter's Case 10 in his book *Asthma*: this case is classical of the troubles and tribulations of any child possessed of an inborn bronchiolar diathesis to hypertony and its susceptibilities to clinical asthma, or 'a tightness of breathing with wheezing on taking a cold which leads to cough and mucus'. That the 'trigger' may vary from time to time in the same child is recorded in Case 10, where not only was acute infection a trigger but occasionally 'the proximity of a cat' or a seasonal 'hay' episode would have similar bronchiolar effect.

Salter stated in the following table his views on 'agents or circumstances tending to provoke the asthmatic paroxysm':

Provocatives of Asthmatic Paroxysm.	Affecting the air-tubes primarily and directly. *Intrinsic*	Things inhaled . . .	Particular kinds of air.
			Chemical and mechanical irritants.
			Animal emanations.
		Inflammation of air-passages	
		Offending condition of blood . . .	After *all* food.
			After certain ingesta, as wine.
			From dyspepsia.
	Applied to some remote part. *Extrinsic*	Reflex nervous irritation . . .	Through the organic nervous system.
			Through the cerebro-spinal nervous system.
		Central nervous system . . .	Irritant psychical.
			Irritant physical.

Henry Hyde Salter (1823–71), M.D., F.R.C.P., F.R.S., one-time professor of physiology and later physician to Charing Cross Hospital Medical School, London, was noted as an eminent clinical teacher who 'spent much of his professional life studying that strange capricious malady asthma', a disease which had afflicted him since his own childhood.

Salter wrote on blood-cells and on the macroscopic anatomy and the histological structure of various parts of the cardiovascular system. His written work was always 'lucid in arrangement and very graphic in description', and he was said to be 'painstaking as a teacher, endowed with considerable command of language and great readiness in illustrative drawing' (*Lancet*, 1871, **ii**, 415). In his own book on *Asthma*: *its Pathology and Treatment* (1860) Salter pleaded for more knowledge on the

> phenomena and laws of that particular form of perverted nervous action in which asthma essentially consists . . . the importance of correct pathological views in relation to the therapeutics . . . and a desire to refute the various erroneous theories that have been advanced with regard to the pathology of asthma and to supplant them by something precise and definite.

It was written in his obituary (*Brit. med. J.*, 1871, **ii**, 278) that 'his unobtrusive merit deserves to be commemorated'.

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