Original Communications.

THE CONSEQUENCES OF ASTHMA.

By HYDE SALTER, M.D., F.R.S., Fellow of the Royal College of Physicians, and Assistant-Physician to Charing Cross Hospital.

Tendency of Asthma to disorganise. The Connsequences of Asthma fourfold: 1. Its direct results on the Bronchial Tubes; Hypertrophy of the Bronchial Muscle; Bronchial Contraction. 2. Results of Obstructed Pulmonary Circulation; *a*, In the Lungs (Congestion, Œdema, etc.); *b*, In the Heart (Hypertrophy, Dilatation); *c*, in the Systemic Venous System (Venous Stasis, Œdema, etc.). 3. Emphysema; Is pure Asthma capable of generating it? 4. Acquisition of the Asthmatic Physique; Its Distinctiveness; Its Characteristic Gait, Physiognomy, and Configuration; Rationale of the Asthmatic Spinal Curvature; Peculiar Chest of Young Asthmatics.

Asthma never kills; at least, I have never seen a case in which a paroxysm proved fatal. If death did take place from asthma, it would be by slow asphyxia—by the circulation of imperfectly decarbonised blood; and before this occurred I think the spasm would yield. When asthma terminates fatally it does so by the production of certain organic changes in the heart and lungs; and it is on this tendency to the generation of organic disease that the gravity of asthma depends.

The consequences of asthma admirably illustrate two laws of our organisation: one, that the workings and processes of life are so intimately bound together, so exactly fit and interlock, that one cannot go wrong without dragging the rest with it; the other, that healthy function is as necessary to healthy structure, as healthy structure is to healthy function. Without asserting that the perverted function of a tissue or organ is in all cases dependent upon some real though perhaps inappreciable perversion of its structure or constitution (which, though perhaps probable, is at present beyond our demonstration), we may safely affirm the converse—that no tissue or organ can long be the seat of perverted action, without perversion in its structure or constitution inevitably following. Organs are made for action, not existence; they are made to *work*, not to *be*; and only when they *work* well can they *be* well. It is the universal law of organisation, that the function of parts shall be, if not absolutely coincident, at any rate indissolubly connected, with their nutrition. The very nutrition of organs is planned on the supposition of their being working machines, and in exact accordance with the work they have to do; so that their working can neither be suspended nor deranged without interfering with their nutrition, and therefore with their structure. Organs either misused or disused invariably organically degenerate.

If we examine the chest of an asthmatic who has but recently been affected with his disease, or whose attacks have been infrequent, we shall very likely find evidence of perfect anatomical soundness of all its organs; but if we examine him again in ten years we shall to a certainty, if the patient has in the interval suffered constantly from attacks of his malady, find evidence of organic disease of the lungs, and very likely of the heart. Now, why is this? Why should organic disease be the inevitable sequel of that which is at first a mere functional and occasional derangement? From the very law I enunciated just now—that functional disorder cannot exist without dragging in its train organic change.

The consequences of asthma appear to me to be fourfold:—1. The direct results on the bronchial tubes themselves of the inordinate action of their walls; 2. All those results of obstructed circulation, first pulmonary, then systemic, which the inadequate supply of air to the lungs induces; 3. That special result of the unequal and partial distribution of air to the lungs—emphysema; 4. The general effect of the disease on the physiognomy and build of the patient—the production of what may be called the *asthmatic physique*.

1. *Direct Results of the Asthmatic Spasm on the Bronchial Tubes themselves.* Organic muscle obeys the same laws as voluntary; its nutrition, and therefore its development, is proportioned to its activity; and this evidently from the same final cause—that it may be equal to its work. No sooner is its activity exalted, or more work thrown upon it, than it immediately hypertrophies: witness the urinary bladder in stricture, and the gall-bladder in biliary calculus. The bronchial tubes in asthma afford but another example of the same thing, and their excessive action issues, from the operation of the same law, in a similar hypertrophous development. Accordingly, we constantly find, amongst the morbid appearances mentioned in the *post mortem* examinations of asthmatics, an undue conspicuousness and thickening of the circular fibres of the bronchiæ. One certain result of this hypertrophy of the bronchial muscles is a permanent thickening of their walls, and consequent narrowing of their calibre; and one possible result is a greater disposition on the part of the hypertrophied muscle to take on a state of contraction. To the former, perhaps, is in part due that slight constant dyspnœa which is so disposed to develope itself in asthma; to the latter, the increased tendency to, and frequency of, spasm which characterises some cases.

Contraction of the bronchial tubes—a permanent diminution of their calibre—is another direct result of asthma. A certain amount of narrowing is inevitably involved in the increased thickness of their walls, due in part to the hypertrophy of their muscular element, in part to the congestive tumidity of the mucous membrane, which is the almost invariable accompaniment of long continued asthma. Perhaps, too, as suggested by Dr. Williams, the very fact of an increase of the contractile element of the walls of the bronchiæ involves a degree of permanent passive contraction in excess of what is natural, just as the irritable bladder, is a contracted bladder as well as a thickened one. A certain amount, then, of thickening, and a certain amount of contraction of the bronchial tubes, is fairly to be assigned to asthma, and has in it its sole and sufficient cause.

But we sometimes find in cases of asthma a degree of contraction far beyond what is thus explicable, amounting to complete occlusion. Now remembering how commonly asthma is complicated with bronchitis, how exactly such a condition is that which old severe bronchitis tends to produce, and how inadequate simple spasm seems to produce it, I am inclined to think that the asthma has nothing to do with it, that it is to be assigned wholly to the bronchitis that has complicated the case, and that cases of asthma in which it is found are always mixed cases. This kind of bronchial contraction, converting the tube into a fibrous impervious cord or band, thickened and knotty, I believe to be always inflammatory in its origin.

Dilatation of the bronchial tubes is another morbid condition that has been found in asthma. I think the most dilated bronchial tubes I have ever known were in a case of asthma. But whenever I have seen it there has been bronchitis as well as asthma, and, for the reason I have just assigned in the case of contraction, I should attribute this also to the bronchitis and not to the asthma. I do not see how bronchial spasm could possibly generate it, whereas I do see in the destruction of the vital and physical properties of the bronchial walls by severe inflammation the most rational explanation of its production.

And I may here remark in passing, that there are two circumstances that greatly impair the value and reliability of the specimens of the morbid anatomy of asthma found in our museums:—one is, the looseness with which the word asthma is, and still more has been, used; the other, the extreme frequency with which, even in cases of true asthma, bronchitis has at some time or other existed.

2. *The Results of Obstructed Circulation induced by Asthma.* Asthma is a state of partial asphyxia, and it therefore gives rise to an identical morbid anatomy, differing only from that of absolute asphyxia in its incompleteness, and in those ulterior changes for which, in absolute asphyxia suddenly induced, there is no time. When a man is drowned we know that an impassable obstruction is at once established to the passage of the blood through the capillaries of his lungs; within a minute or two the stoppage is complete. The pulmonary vessels, still filled from behind, become engorged with the blood that they cannot pass on; the right chambers of the heart become distended with the blood which they cannot empty into the engorged lungs; and thus, the obstructing force propagated backwards, the cavæ and their tributaries become distended with accumulated blood in increasing quantities as long as life is prolonged. Meantime, the left side of the heart receives hardly any blood, and its action fails for want of its normal stimulus, while the

right side becomes less and less able to contract on its bulky contents.

Soon, from these opposite causes, the heart ceases to beat, and life is extinct. We open the body, and find—arteries, left side of the heart, and pulmonary veins empty; pulmonary artery, right side of the heart, and all the great veins gorged with black blood. Now there can be no doubt that exactly the same thing takes place at every attack of asthma, but only to a degree that is compatible with life. If we could see into an asthmatic during a fit we should see a certain dose of the same deranged distribution of blood, and from the same cause—pulmonary capillary arrest from the shutting off of air. We should see pulmonary venous congestion, distended right heart, large veins full, and a scanty supply of arterialised blood finding its way to the left ventricle. All external manifestations are consistent with this:—the small and feeble pulse, the irregular and faltering systole, the turgid veins of the head and neck, the occasional hæmoptysis, the dusky skin. If all the bronchial tubes could be simultaneously so contracted as to be completely occluded the same result would take place as if a ligature were placed round the wind-pipe, the deprivation of air would be complete, and death would supervene. But such is not the case, all the bronchiæ are not simultaneously contracted, and their contraction does not amount to complete occlusion. The arterialisation of the blood is lowered, not arrested, and life is maintained. By and by the spasm yields, air is freely admitted, the bar at the capillaries ceases, the pulmonary veins unburden themselves, and all is well again.

But can such a state of things exist long, or exist often, without producing other organic changes? Certainly not. No tissue or organ can be long or often the seat of vascular disturbance without becoming more or less disorganised. I shall consider the changes that result from this stoppage at the pulmonary capillaries that every attack of asthma gives rise to in the order in which they occur,—in the lungs, in the heart, and in the systemic venous system; that is, in those three segments of the circulation along which, in a retrograde direction, the obstructing force is propagated.

In the Lungs the first result is what is called venous congestion—a term in part correct and in part erroneous; for, while the congestion is congestion with venous blood, the congested vessels are really branches of the pulmonary arteries. I am not aware that the exact seat of this congestion is determined, whether it is limited to the branches of the pulmonary arteries, or involves the capillaries as well. That will depend on where the seat of obstruction is. If the capillaries are congested the seat of obstruction must be in front of them; if the capillaries themselves are the seat of the obstruction they cannot be congested—the congestion must be limited behind them. Now I am inclined to think that the exact seat of the obstruction is in the minute venules, just where the blood is passing from the capillaries into the pulmonary veins; and my reasons are these:—The cause of the stoppage is the blood not being what it should, not being properly decarbonised: now, the capillaries are the seat of its decarbonisation; it is not, therefore, until it leaves them and arrives at the ultimate pulmonary venous radicles that it becomes what it should not be—venous blood where it ought to be arterial. That point, then, where its defective arterialisation must be first recognised, must be the point of its arrest. This would imply capillary engorgement. Whether the absolute capillaries are involved in the engorgement I cannot say, or whether it stops at the ultimate twigs of the pulmonary artery. It should be made the subject of careful microscopical observation, which I have not yet made it.

Indeed, I am not aware that the state of the vessels in chronic pulmonary congestion has ever been made the subject of microscopical investigation.

One result of this impeded circulation through the lungs which I believe will one day be demonstrated, is thickening of the walls of the ultimate arterial twigs analogous to that which Dr. Johnson has shown to take place in impeded circulation through the kidney, and produced in an identical way. This too, I regret to say, I have never since the idea occurred to me had an opportunity of verifying. Another result is, that the engorged vessels gradually lose their tone and yield to the distending force of the blood, so that the congestion becomes more and more considerable and of more and more easy induction. Another result is, that the serous portion of the accumulated blood transudes the walls of the vessels, and, escaping into the areolar tissue and air cells, gives rise to œdema. From this accumulation of blood, and displacement of the air in the air cells by serum, parts of the lung may undergo what has been called splenization, becoming quite solid, airless, sinking in water, non-crepitous, and black. And this is the state in which the more dependent parts of the lungs of those who have died of chronic asthma are often found;—a state not to be distinguished from that of the lungs in fatal chronic bronchitis, and which is, in fact, the morbid anatomy of slow asphyxia, however produced.

Hypertrophy, and dilatation of the right side of the heart has long been a well known and recognised complication of asthma. In examining the chest of an asthmatic patient we often find the heart's pulsation plainly felt, and even seen, in the scrobiculus cordis, while in its normal situation it can hardly be perceived. For this there are several reasons. One is, that the violently acting diaphragm draws the heart down lower than usual; another, that an emphysematous left lung may thrust it downwards and to the right, and also, by overlapping it, produce that undue resonance in the region of the heart's dulness and that indistinctness of the apex-beat beneath the nipple which are recognised signs of emphysema in this situation. But a third reason undoubtedly is dilatation of the right ventricle.

Although this last is the only cause I have seen assigned for this displacement of the heart's beat, I insist on the other two as adjuvant, because they are evidently sufficient of themselves to drive the heart down into the scrobiculus, and transfer its pulsation thither without any dilatation of the ventricle. In cases of recent asthma, where there has been no time for dilatation or hypertrophy of the right ventricle to take place, and where, in the intervals of the attacks, the situation of the heart's beat has been perfectly normal, I have felt and even seen this pulsation in the scrobiculus very strongly marked at each fit, coming with the fit and going with the fit. Now hypertrophy and dilatation of the right ventricle are not conditions that can come and go. It is evident, then, that a transference of the heart's pulsations to the scrobiculus may be produced by simple displacement of the organ, without any extension of its right chambers; and therefore, when occurring during a paroxysm of asthma, this sign is not to be relied upon as evidence that the heart has organically suffered.*

But why should heart-disease be the legitimate sequel of asthma? Why should the stricture of the bronchial tubes tend to produce hypertrophy and dilatation of the right ventricle? The connection of these remote and apparently dissociated conditions is at once supplied by the law of asphyxia which I have just now referred to—that the shutting off of air from the lungs immediately brings the pulmonary circulation to a stand-still, and places in front of the right chambers of the heart an obstacle which they cannot overcome. This obstacle at the pulmonary capillaries provokes unwonted efforts on the part of the right ventricle, which of course becomes more or less hypertrophied. After a time the ventricle yields to the distending force of the accumulated blood, next, the auricle, and finally, the great veins and the whole of the venous system; so that ultimately asthma may end in general venous congestion and dropsy, just in the same way as primary cardiac disease. But asthma may go on for a long time without any such results. It is surprising how severe the paroxysms may be, and for how many years the disease may continue (provided the fits are not prolonged and frequent), without the heart being in the least affected. It is only in cases of very long standing, and when the fits are tedious, and leave a certain amount of permanent dyspnœa in the intervals (especially if there is some bronchitic complication), that these changes in the heart take place. As far as my experience goes, I should say that they never occurred as long as the recovery in the intervals was absolute. I cannot therefore agree with Dr. Todd (*Medical Gazette*, vol. 46, p. 1001) in the importance he assigns to evidence of dilatation of the right ventricle as a diagnostic sign of asthma. "I look upon this sign," he says, "as one of the most characteristic symptoms of asthma; and I consider its presence in any case where I suspect asthma as a clear confirmation of the correctness of those suspicions. In accordance with this view, one of my first steps in examining a patient whom I suspect to be asthmatic is to apply my finger to the scrobiculus cordis. If I find no beating of the heart there, my conclusion is a contingent negative. But if I find it beating there, and not in its natural

* Of the truth of this, any one may satisfy himself by placing his finger on his scrobulus cordis, and taking a deep inspiration, when he will immediately feel the cardiac pulsations, which will continue as long as he keeps his chest at full distension;—he expires, and it is gone. Here you have the same conditions with regard to the situation of the heart that you have in asthma, only in a less degree—lung distension and flattening of the diaphragm; in fact, in asthma the parietes of the chest, diaphragmatic and costal, are in a state of permanent extreme inspiration.

position under the nipple, my conclusion is a certain affirmative."

Doubtless, in a case of suspected asthma, evidence of dilatation of the right side of the heart would strengthen the diagnosis; but the absence of it would go no way at all to negate the supposition that asthma existed. It would simply show that one of the results of asthma had not yet arisen, and it would establish a presumption that the disease had not been of long standing, that it was unassociated with chronic bronchitis, and that it had as yet inflicted no organic changes on the lungs. A patient's heart-beat may be in every way normal, and yet half an hour ago he may have been in the agonies of an asthmatic paroxysm. The positive evidence of heart-change in asthma is of some value; its negative evidence is worthless.

3. *Emphysema* is certainly the commonest of all the morbid changes that asthma tends to produce. I should say it was extremely rare to find the lungs of those who have suffered from asthma long entirely free from emphysematous inflation. The best examples of emphysema that I have ever seen have been in the *post mortem* examinations of chronic asthma.

Adopting that view of emphysema so ably advocated by Dr. Gairdner—that it is essentially a compensatory dilatation, and implies the neighbourhood of non-expansible lung—I believe the mechanism of the production of emphysema by asthma to be as follows:—The bronchial spasm shuts off the air; the shutting off the air produces capillary stasis—partial asphyxia; the congested vessels relieve themselves by the characteristic mucous exudation; the continued occlusion of the bronchial tubes, if the spasm does not yield, shuts up this mucus, and prevents its escape, and at the same time, by barring the access of air, prevents efficient cough; so long as the spasm lasts, therefore, its escape is doubly prevented—by direct obstruction, and by the want of the natural machinery for its expulsion. The tubes affected by the asthmatic contraction thus become doubly obstructed—at first narrowed by spasm, and then completely occluded by mucous infarction. As long as the spasm lasts the escape of the mucus is impossible. In the meantime, whatever may have been the length of the attack (and we know that it often lasts for days), the inspiratory muscles are making the most violent efforts to fill the chest, and are, in fact, keeping it in a state of extreme distension.

The length of time required for the removal of air from a lobule, from which communication with the external atmosphere is completely shut off by occlusion of its corresponding bronchial tube, I do not know; so I do not know if, in an attack of asthma, any actual lobular collapse could take place, although, in a prolonged attack of some days I feel no doubt that it would. At any rate, the lobules whose bronchiæ are occluded cannot yield to the distending force of the inspiratory muscles; the whole distension of inspiration is, therefore, spent on those portions of the lungs whose communication with the external air is free; the open lobules have to expand for themselves and their occluded neighbours, and undergo an excessive inflation in proportion to the amount of lung that is non-expansible,—in other words, become emphysematous. If we consider how complete the occlusion must be by this double process of spasm and infarction, how protracted asthma often is, and how violent are the inspiratory efforts that characterise it, I do not think we shall wonder at any amount of emphysema that is thereby produced, nor at its being one of the commonest organic changes to which asthma gives rise.

It will be seen, from the account I have just given, that asthma produces emphysema just in the same way that bronchitis does. The two processes are essentially identical. In the one case the bronchial tubes are narrowed by inflammatory thickening of their walls and occluded by inflammatory exudation (muco-pus); in the other, they are narrowed by spasmodic contraction and occluded by the exudation of congestion (viscid mucus). The only difference is, that the narrowing and occlusion of bronchitis are generally of longer duration than those of asthma.

I think Dr. Walshe is quite right in his opinion that "The connection of emphysema with spasmodic seizures is certainly sometimes, possibly always, dependent on an intervening irritative or passive congestion of the tubes." That it is so sometimes I think is certain, because I think that congestion of the tubes is the immediate result of prolonged spasm. But is it always? If not, then emphysema may result from the spastic occlusion of the tubes without any mucous infarction. Is this possible? Is asthmatic spasm ever so complete and continuous as to produce the results of plugging,—lobular isolation, and collapse? This is a question that I think would be very difficult, at present perhaps impossible, to answer. One thing I feel strongly persuaded of, that it is not necessary that there should be any bronchitic complication, anything actually inflammatory, in order that asthma should result in emphysema. I have seen emphysema developed in a case of asthma in which bronchitis never existed.

It is hardly worth while for me to describe the symptoms that mark the closing scene of those miserable cases of asthma that terminate in the production of these organic changes in the heart and lungs that I have just been describing. When once the right cavities of the heart have become dilated and the obstructing force retrogrades upon the systemic veins, the symptoms are not to be distinguished from those which characterise a similar condition induced by chronic bronchitis. The cases differ alone in their previous history. There is the same rattling wheeze, the same choking cough, the same orthopnœa, the same abundant frothy expectoration (but in the case of bronchitis more purulent), the same venous regurgitation, the same choked-up breathlessness, getting ever worse and worse as the œdema and congestive solidification rise higher and higher in the lungs, the same general œdema beginning at the feet and gradually creeping up the trunk, the same cyanosis. The sufferings of this gradual choking-out of life are most painful to witness till the increasing heaviness from the circulation of venous blood in the brain deepens into the insensibility which ushers in dissolution.

OBSERVATIONS ON SOME OF THE OBSCURE FORMS AND VARIETIES OF AGUE: WITH GENERAL REMARKS ON THE DISEASE.

By E. GARRAWAY, Esq., Faversham.

[*Read before the East Kent and Canterbury Medical Society.*]

THE phenomena of ague, so characteristic, so well known, and so often witnessed in this division of the county, might well be supposed to afford no difficulties in diagnosis; and when developed in its three consecutive stages of cold, fever, sweat, followed by immediate recovery, there can be none; but the variations which these phenomena assume, their sometimes absence, and their not unfrequent substitution by other symptoms, apparently totally unconnected with ague, and only known to be in alliance with it by their remission and return, render its discrimination a matter occasionally of no slight embarrassment.

You are sent for to a young man in previously good health, who is suddenly seized with great pain in the side, accompanied by severe vomiting, with or without acceleration of pulse, according to the time he may have been under the influence of the attack. You discard the idea of any inflammatory affection, and have little hesitation in pronouncing the case one of passing gall-stones. Nothing of the kind! Wait awhile, and your patient breaks out into a profuse sweat, and in a few hours is well; only, however, to have a repetition of the seizure on the next day or the day following.

A mother sends in terror for you to see her infant, perhaps only three or four weeks old. The child is cold; its extremities are blue; pulse almost imperceptible;—in short, the little creature appears moribund. You think of imperfectly closed foramen ovale, of internal hæmorrhage, of accidental poisoning,—you do not know what to think, and communicate your fears to the anxious parent. Needless alarm! In due course the infant becomes warm again, then hot, and finally covered with beads of perspiration. This also was ague.

You are called to a child in convulsions, or to a supposed case of brain fever; perhaps, a young woman, whom you find delirious, with quick pulse, dry tongue, and burning skin. You are strongly tempted to leech, blister, and adopt antiphlogistic treatment; but, on inquiring, find that two days preceding the young woman or the child was similarly affected, though possibly in a lesser degree. This is all-sufficient for your diagnosis, you predict a resolution by sweat, and in a few hours your patient is recovering for that time.

The next case you find in bed, with a burning skin, rapid pulse, parched brown tongue, every limb racked with pain,—in short, all the symptoms of a low, continued fever. Such you pronounce it, and order aperients and salines. To your astonishment, on calling the following day, you find your patient eating his dinner, with a moist, clean tongue, cool skin, quiet pulse; in fine, convalescent. You rejoice with him on his speedy recovery. Vain exultation! To-morrow he is worse than ever;