A Clinical Lecture

ON

PAROXYSMAL DYSPNOEA IN CARDIO-RENAL PATIENTS:

WITH SPECIAL REFERENCE TO "CARDIAC" AND
"URAEMIC" ASTHMA.

DELIVERED AT UNIVERSITY COLLEGE HOSPITAL, LONDON,
NOVEMBER 12TH, 1913.

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DURING the past few years interest in the question of dyspnoea has awakened very considerably. I propose at the present time to relate some observations made at the hospital during the past twelve months, which, so it seems to me, help us to a fuller understanding of breathlessness in a large group of patients who have evident signs of cardiac disease. I will first direct attention to a special type of breathlessness from which patients suffer, indicate how far we can explain this breathlessness, and how it may be recognized. Finally, I shall briefly discuss the nature and relations of "cardiac" and "uraemic" asthma, so-called, and suggest certain lines of thought and procedure, which may, I think, bring us into closer touch with the realities of disease in these patients.

A SPECIAL FORM OF DYSPNOEA.

First I would direct your thoughts to instances of simple cardiac dyspnoea. There are forms of congenital heart disease and of extreme mitral stenosis in which, as you come to the bedside, you immediately recognize a deficiency in the aëration of the arterial blood. The patients I would have you call primarily to mind are those in whom you have seen deep or extreme cyanosis. Some of these patients are very breathless, but it is notable that in many others, especially those who suffer from congenital heart defects, the anxiety of breathing is not severe while they lie at rest. From such observations I argue that if dyspnoea is due to deficiency in the aëration of the arterial blood, cyanosis will be prominent, and, by a comparison of many cases, that urgent dyspnoea, if of the same origin, must be accompanied by extreme discoloration. Next I would point out that a very large proportion of patients who exhibit great anxiety of breathing and have evident signs of cardiac disease, do not exhibit this degree of cyanosis. It is a well-known fact that in heart disease breathlessness and cyanosis are often shown in disproportionate degree, yet the significance of this fact has, I think, escaped attention. Why it has so escaped I shall attempt to explain later. At present let us be content with the observation and the natural conclusions to be drawn from it. If a patient is urgently breathless and an equivalent cyanosis is not found, the dyspnoea is not wholly due to deficient aëration of the blood. You can rarely pass through the wards of this hospital without seeing a number of patients sitting propped up with pillows and in great distress of breathing; examine these cases more closely and you will see that some of them have but little cyanosis. These patients belong to the special group which I shall now describe, and for descriptive purposes I shall give the relevant features of an extreme case.

In the early months of this year I saw a man, aged 57, who for twelve months had experienced failing health. Breathlessness was his chief complaint, and it was periodic in its intensity. For months he would be well, or comparatively so, then for weeks his breathing would be difficult, so that active movement was impossible, and he would lie up. During the periods when he was in bed he was continually breathless, and he dreaded the nights because his breathlessness increased during them; often he was awakened from his sleep by attacks of aggravated breathlessness, in which he feared suffocation; these attacks would last a half-hour or more, and he would relapse into a short sleep to be similarly disturbed. When I saw this patient it was evening, and on the first day of his taking to bed. He was not orthopnoic, but lay flat in bed; his respirations were deep, noisy, and periodic (Cheyne-Stokes type); he was drowsy, complained of severe headache, and great breathlessness. Yet the mucous membranes of this patient were as pink as

those of a child; neither in the lips, tongue, ears, cheeks, or fingers, was there the faintest trace of cyanosis. Upon further examination the heart was found to be a little enlarged, the pulse was at the rate of 92 per minute, occasional extrasystoles interrupted its rhythm, and alternation in the force of the pulse beats was seen. There were no murmurs. The veins pulsated freely and seemed a little engorged, but the liver was not enlarged and there was no dropsy. The blood pressure was 200 mm. Hg. He was passing from 600 to 2,000 c.cm. of urine a day; sometimes a trace of albumin was present, and a few granular casts were found. Over the lungs a few rhonchi were audible.

I take this as a type case and emphasize the following features. The man presented some signs of a degenerate heart muscle, enlargement, and especially pulsus alternans; he presented signs of renal involvement, the urine flow was, as a rule, increased, casts were present, the blood pressure was very high. He had urgent dyspnoea, yet the blood was perfectly aërated. You may say that I am describing a case of so-called "uraemic" dyspnoea; I cannot disagree with you. This patient was one of a series specially investigated for me by my collaborators, Mr. Barcroft, Drs. Ryffel, Cotton, and Wolff. Mr. Barcroft found in him by special methods evidences of a diminished alkalinity of the blood sufficient to account for the dyspnoea and due, not to CO₂, but to non-volatile acids. Having studied a number of cases by these methods, which I will describe briefly later, we were able to isolate a group of symptoms which are specially associated with an altered blood reaction of the kind.

Special Clinical Associations of Relative Acidosis.

The most important feature of these cases is the breathlessness, varying in degree, as Mr. Barcroft has shown, with the reaction of the blood, being of the periodic type known as Cheyne-Stokes respiration, being accompanied when severe by conspicuous restlessness during the periods of hyperpnoea, and lastly becoming exaggerated by the slightest exercise. There is no reserve, the breath cannot be held, and forced breathing is followed by no apnoeic period. Oxygen affords little or no relief, though it alters the type of breathing. Secondly, there is evidence that the blood is well or moderately aërated; there is no cyanosis comparable with the dyspnoea. Thirdly, the dyspnoea is more severe towards evening and in the night, and sleep is broken by exaggerated attacks of breathlessness. Fourthly, the pulse-rate is increased (80-100), and especially towards evening. Fifthly, the temperature is subnormal.

These are almost constant clinical manifestations; but the picture may be modified in a variety of ways, and it is to these modifications that the chief difficulties in diagnosis and the confused pathology of the past are due. Imagine a patient with the symptoms which I have described and take him as a type; add to this symptom group the symptoms and signs of heart disease in its varying forms; add to it also a large number of unquestionable renal manifestations; add to it emphysema and the symptoms of cerebral arterial sclerosis, and you are able to construct all the different types in which this curious form of acidosis is found. The types are protean. Your power of diagnosing acidosis will depend on your ability to discriminate the underlying symptom group which I have detailed. Usually you will have no difficulty; the condition is an extremely common one, and urgent dyspnoea, where there is a want of equivalent cyanosis, is your first guide; really distressed breathing in elderly subjects, accompanied or not by cyanosis, is always open to suspicion; the presence of Cheyne-Stokes breathing, and especially the characteristic nocturnal attacks, are extremely valuable indications. The periodicity of the breathing is often inconspicuous or variable; it may consist merely of slight waxing and waning in the amplitude of the breathing, there may be no true apnoeic periods; it may be present only at night or at the patient's first admission; it is very often overlooked; I have heard its presence denied in patient after patient, and have found it either by immediate close examination or by repeated examination. It should be looked for especially when the patient is quiet or asleep; very small disturbances may abolish it; if you watch your patient when he is unaware that you are watching you may often detect it. But Cheyne-Stokes breathing may not be present; it may be absent in at least two types of case—(1) where the

relative acidity is slight, and (2) where moderate cyanosis is added to the picture, as it may be.*

The evening or nocturnal attacks may be of a few minutes' duration; they may last an hour or more; they give rise, when severe, to the most urgent distress, all the accessory muscles of respiration being called into action. The patient sits clutching a bed-rest or the arms of a chair, and literally gasps for air. The agony is often intense; he may become delirious.

Finally you have the pulse-rate and temperature chart to help you. But in regard to the first, this evidence is obscured where rapidity and irregularity of the ventricle are due to fibrillation of the auricle. And the temperature may not be subnormal, it may be at or above normal. A rise from a subnormal position to the normal in the absence of improved breathing is to be regarded in these patients as equivalent to a raised temperature in other patients.

Further Associations.

I have isolated the foregoing symptoms because they are what I regard as the essential symptoms. I now propose to consider the remaining symptoms and signs which are common accompaniments. There are first of all symptoms and signs of renal disease. The urine is increased in quantity and nocturnal micturition is common; it is of low specific gravity, and may or may not contain a trace or cloud of albumen; casts are usually seen in centrifugalized specimens, but may not be discovered. Severe headache, vomiting, twitching of the limbs, temporary aphasia or hemiplegia and convulsive attacks are not infrequent. Retinal changes, oedema of renal distribution† and haemorrhages have not been observed as yet. High systolic blood pressure (190 to 220) is very frequent, and the arteries are almost always tortuous or thickened. Wasting is very common, in fact the rule; anaemia of slight grade is also frequent, the red cells numbering three million and a few hundred thousand or more.‡ Inflammatory changes in the pleurae are often seen in severe cases; unilateral effusions are particularly prone to develop and often develop suddenly.

Equally important are the symptoms and signs of heart disease. The heart shows signs of enlargement; dilatation and hypertrophy may be very considerable. Often there are no murmurs, but gallop rhythm is heard at the apex. This is the type of case in which renal heart is customarily diagnosed. On the other hand, aortic regurgitation, mitral stenosis, or mitral regurgitation often accompany the intoxication, and I have seen aortic stenosis and aneurysm of the arch. In several cases of our series there was a history of rheumatic fever. The mechanism of the heart may be disturbed in any known way. Fibrillation of the auricle and alternation are the commonest forms of disturbance, but extrasystoles, heart-block, paroxysmal tachycardia have also been seen. Certain of these disturbances of rhythm mask or hide the acceleration of the normal heart rhythm. Anginal symptoms are not infrequent, and may be severe. Lastly, circulatory failure may show itself in venous engorgement, cyanosis, and dropsy.

The Influences of Venous Engorgement.

I have spoken of a type case in which urgent dyspnoea was unassociated with the slightest cyanosis, but this combination is rare, because practically all these patients have cardio-vascular trouble in greater or lesser degree. When, as is commonly the case, venous engorgement supervenes and the liver swells, the patient shows cyanosis, which may be slight, moderate, or deep, according to the degree of embarrassment. With the addition of stasis—and it is nearly always present in some degree—the patient early assumes the orthopnoeic position. The special cause of dyspnoea—the presence of non-volatile acids in the blood stream—has the cause of simple cardiac dyspnoea super-added to it—namely, a want of aeration of the blood. The circulatory embarrassment hides the underlying condition more or less completely, and the whole dyspnoea is, as

a consequence, often assigned to the heart alone, or to the heart and lungs, because bronchitis, oedema of the bases, and pleural effusions may develop. The diagnosis is sometimes impossible for these reasons; a very careful comparison of the degree of dyspnoea and the degree of cyanosis is always called for. The nocturnal attacks and the subnormal temperature are the most valuable signs in the complicated cases; Cheyne-Stokes breathing also occurs in many of them, though in some where the cyanosis is more noticeable it may not be seen. The paroxysmal breathlessness may be replaced by persistent or urgent breathlessness under similar circumstances. The stasis also masks signs of renal involvement; the quantity of urine is diminished and falls below normal, it becomes of high colour, and contains excess of urates and urobilin; albumin and casts are then mistakenly attributed to renal congestion alone. The characters of the urine in the condition are therefore inconstant; I believe they are inconstant simply because stasis so often modifies them.

Contrast the patient I have already spoken of with the following:

A wasted man of 67 years who is propped in the orthopnoeic position. The facies are dark, the lips cyanosed; the ears, the tongue, and the fingers are more deeply cyanosed. The breathing is gasping and wheezy, and is heard distinctly over the whole ward. He is in great distress, the muscles of the shoulders tense, and the chest is moved rigidly. As he is watched, the distress passes, and he sinks back in a drowsy condition as the respirations fade away. He becomes apnoeic and for the moment is still, but the breathing soon starts again, and as it grows in amplitude the restiveness and distress return. He has been ill for several years; seriously ill for six months. When examined, the heart is found to be greatly enlarged, dilated, and hypertrophied; systolic murmurs are audible at the apex and aorta. The auricles are fibrillating, the pulse grossly irregular. The arterial tension is high and the vessels are thickened. The veins are engorged and pulsate freely. The liver is much enlarged and pulsatile. There is anasarca of the lower limbs and trunk. There are signs of ascites and small effusions into both pleurae. The urine is high-coloured and heavy; it is scanty and contains albumin, pus cells, and casts.

The history tells us of attacks of suffocation at night, and he still has them. The temperature stands at 98°.

This patient had a great excess of non-volatile acids in the blood, sufficient to account for most of his dyspnoea. He was cyanosed, and had abundant evidences of heart failure with pleural effusions and bronchitis. The renal signs were less conspicuous.§ The cause of the dyspnoea was the same as that in the first case I have described, but the clinical picture was profoundly modified by the addition of cardiac failure.

I might cite similar cases; or instance those in whom aortic disease, mitral stenosis, forms of heart irregularity other than auricular fibrillation or angina pectoris were prominent, or in whom Cheyne-Stokes breathing was never seen. Such cases are commonly relegated to the category of "cardiac asthma," because, having conspicuous signs of heart disease, they suffer from paroxysmal breathlessness, especially severe at night.

I might instance, on the other hand, those with simple cardiac enlargement and gallop rhythm, or those in whom persistent drowsiness, headache, vomiting, twitching, or actual convulsions were prominent features; or in whom a retention of urica was discovered; these cases are usually spoken of as "uraemic asthmatics." We find the same blood picture in association with all these symptoms and signs, and attribute the dyspnoea, or the greater part of it, to a single and constant cause—namely, a diminished alkalinity of the blood from the presence of non-volatile acids.

Morbid Anatomy.

A large percentage of our cases have come to *post-mortem* examination. The morbid anatomy is as equally varied as the symptomatology. Hypertrophy and dilatation of the heart is the rule, though it varies in degree; the heart and vessels may weigh anything from 15 to 25 ounces. Coronary arterial disease is almost constant; fatty change has been seen in several of our cases;¶ valve lesions of all kinds may be present. The kidneys have always shown some change; in a few they were of

* It will be remembered that Pembrey found that Cheyne-Stokes breathing is abolished when CO₂ is inhaled. Most of his cases were of the type now described. (*Proc. Physiol. Soc.*, February 24th, 1906.)

† Acidosis has since been found by Mr. Barcroft in a young man with all the symptoms of chronic parenchymatous nephritis, including universal oedema.

‡ A leucocytosis (13,000 to 19,000 per c cm.) is not uncommon; the increase is mainly in the polymorpho-neutrophile cells.

§ At the *post-mortem* examination advanced granular contracted kidneys were discovered.

¶ A number of cases are described in a recent article: Lewis, Ryffel, Wolf, Cotton, and Barcroft. (*Heart*, 1913, v. 45.)

¶ It was noted by Stokes in association with cases of a very similar type.

the advanced granular contracted or arterio-sclerotic type; more commonly they were large and showed diffuse fibrosis and lymphocytosis with changes in the medial and internal coats of the smaller vessels. General atherosclerosis of the larger vessels was usually discovered.

The Immediate Cause of the Dyspnoea.

The diminished alkalinity of the blood has been identified for us in these cases by Mr. Barcroft.* He exposes a sample of whipped and aerated blood to a definite pressure of oxygen, and finds that, according to the degree of its alkalinity, it absorbs more oxygen. The test of the reaction is the degree of saturation of the haemoglobin with oxygen when exposed to a definite pressure. The test is a delicate one; if the quantity of oxygen absorbed is known, the amount of acid which must be added to normal blood to produce a similar reaction may be calculated in percentage terms. If the same blood is exposed to oxygen, in the presence of CO₂ at pressures equivalent to those found in the alveolar air of the patient from whom such blood was taken, you have, according to the amount of oxygen absorbed, a measure of the reaction of the blood as it circulates in the patient. You gauge in this manner the total relative acidity (or hydrogen-ion-concentration) and this was found in our patients to give an accurate measure of the degree of breathlessness established in any given case. Not only so, but the results of this second test varied with the degree of breathlessness in the same case from time to time; so that when improvement in breathing was noted clinically the reaction of the reconstructed blood became more alkaline; or when the dyspnoea became more urgent, the blood was found to have greater acidity. Mr. Barcroft, working at Cambridge, was almost as well aware of changes in the degree of breathlessness of our patients in this hospital, as we were here, and by the simple examination of blood and alveolar air samples sent to him.

Any increase in the acidity of the blood increases the depth and rate of respiration. This fact has been abundantly proved; and it is a matter of indifference if the excess of acid is volatile, as it is when CO₂ is breathed, or whether it is non-volatile. There are many notable examples of the latter. I may instance the experiments of Leymann,¹ who introduced small quantities of phosphoric and other acids into the circulation. The injection of acids produces hyperpnoea, of alkalis apnoea. The dyspnoea of violent exercise is due to accumulated lactic acid.² Diabetic asthma is the result of diacetic and beta-oxybutyric acids. The presence of excess of non-volatile acid stimulates the respiratory centre, and hyperpnoea results; the hyperpnoea washes out CO₂, it increases the pressure of oxygen in the alveoli, and in this way the body attempts to accommodate itself to the altered conditions; but the accommodation is imperfect, and the reserve is decreased or abolished. The fall in the pressure of CO₂ in the alveolar air produced in this fashion is noteworthy; it serves as an index of altered respiratory metabolism, and suggests the presence of acidosis. The alveolar CO₂ may be reduced in exercise (Cook and Pembrey); it is diminished in diabetes³; it was reduced in our cases where it was estimated. It was a diminution of alveolar CO₂ pressure and a lowering of the respiratory quotient which led Straub and Schlayer⁴ recently to suggest acidosis as the cause of breathlessness in cases they term uraemic; the cases they cite are certainly of the same group which I have described to you. As to the nature of the acid in the cases investigated we have no certain knowledge. In two moribund cases an excess of lactic acid was found by Dr. Ryffel, but as a rule it could not be identified. A similar acidity is developed by normal subjects at high altitudes, and the symptoms accompanying this relative acidity are similar to those which our patients exhibit; Cheyne-Stokes breathing and hyperpnoea, intensified at night, are common phenomena in those who climb to such heights as ten or fifteen thousand feet. Sufficient for the time being that relative acidosis is present, and that it is the cause of dyspnoea in a very large number of the patients of our wards. Moreover, it seems possible, if not probable, that in many of these cases it is also responsible for the increased pulse frequency and loss of cardiac tone, expressed as dilatation.

* For further details of the method the reader is referred to our full paper, *Heart*, 1913, v. 45.

CARDIAC AND RENAL ASTHMA.

We distinguish between "cardiac" and "renal" asthma to-day in one of two ways. First, by studying the associated symptoms; if there are other "uraemic" manifestations, for instance, the curious ethereal breath, twitchings of the muscles, drowsiness or vomiting, a renal asthma is diagnosed. Each of these symptoms may occur in combination with the special type of breathlessness which I have described, namely, the breathlessness associated with acidosis; on the other hand, each and all may be absent in cases of the same group. These symptoms, therefore, are simple associations; they have not the same underlying cause as the asthma. The presence of such symptoms, apart from suggesting that the kidneys are not fulfilling their functions in a given case, does not materially help in elucidating the cause of the asthma; on the other hand, the cardiac symptoms may predominate. Valvular disease, signs of venous engorgement, dropsy, etc., may command our attention. In these cases cardiac asthma is diagnosed.

Secondly, we attempt to distinguish the two types of dyspnoea after death. When the body is examined the conspicuous lesion is found in the kidney, or it is found in the heart; the morbid anatomist points to one or other organ as the seat of the chief mischief, and accordingly the dyspnoea is relegated to the "renal" or "cardiac" category. The answer of the dead body is regarded as decisive; it is probably the least decisive answer we can obtain, and for the reason that no man can say at the present time, from the appearance of heart or of kidney, whether asthma was a clinical feature of the case or not. Neither can the presence or absence of this symptom be judged to-day from the closest inspection of these organs with the microscope. Nothing is clearer than the frequent discrepancy between the degree of functional impairment in these organs and the amount of structural change in them.[†]

We come to this standpoint—that the clinical or anatomical distinction between cardiac and renal asthma is no certain one.[‡] Asthma occurring in patients who show on the one hand prominent cardiac lesions, on the other hand prominent renal lesions, may or may not be due to a single cause.

I would lay especial emphasis upon the failure of morbid anatomy materially to help us; changes of structure may have directed our search; it has not solved, and it never could solve, the problem as a whole. Breathlessness is a symptom during life; the body is examined after it has passed through the process of death, which conceals and eventually buries the truth. We seek the cause of hurried breathing, not in the subject of it, but in his remains, which are incapable of respiration. The chemistry of the dead is not the chemistry of the living. The problem of asthma is to be solved at the time of its occurrence, and it is first a problem of chemistry. While abnormalities of structure fail to indicate the cause, chemical pathology immediately directs us to it. The dyspnoea results, as we have seen, from diminished alkalinity of the blood; appropriate tests applied to the blood of these patients tell us whether these patients are sufferers from dyspnoea, they also reveal the degree of dyspnoea in a very accurate manner.

The object of these remarks is twofold. They are intended to emphasize study in the wards as opposed to that of the deadhouse. They are also made to persuade you that a classification of dyspnoea, based upon structural

[†] English clinicians, as opposed to those of the French school, have too long neglected the chemical side of renal disease in favour of the anatomical.

[‡] I have searched a number of past writings for definitions of "cardiac" and "renal" asthma and for distinctions between the two, but can find no satisfaction in the accounts, when they are regarded from the standpoint of pathology. The terms are used loosely, and in varying senses. The French writers, and Huchard especially, alone seem to have been widely awake to the special meaning of paroxysmal breathlessness associated with Cheyne-Stokes breathing and other characteristic symptoms. Huchard (*Traité clinique des maladies du Cœur*, etc., Paris, 1899, Tome I) gives a perfectly clear account of cases of the same kind as those here described, emphasizing the form of dyspnoea as separate and distinct from simple cardiac dyspnoea, under the term "Dyspnée toxico-alimentaire"; he clearly recognizes that it may be combined with varying degrees of cardiac failure, and that under such circumstances the chief cause of breathlessness is often overlooked. He attributed the dyspnoea to relative impermeability of the kidneys, which incompletely eliminate toxins developed in the alimentary tract. His reasons for this hypothesis are found chiefly in the effects of diet. He thought that the term "aortic asthma" should be replaced by "toxic asthma." His account of asthma is most instructive.

defects, is incomplete and often irrational. I cannot agree with the further use of the terms "uraemic" or "cardiac" asthma, even as terms of convenience; we should speak and think of an asthma associated with relative acidity of the blood, or, more briefly, an asthma of acidosis; we should separate our types of dyspnoea according to the nature of the stimulus which produces it; subdividing those of acid origin according to the type of acid which is in excess, and leaving those in which the same type of acidosis is found, whether such acids can be identified or not, in a single and undivided group, provisionally at all events. Proceeding in this manner we shall immediately separate dyspnoea due to volatile acid (CO_2) and non-volatile acid. In the last group we shall isolate the dyspnoea of diabetes; it results from intoxication by the acids diacetic and β -oxybutyric; and if these acids are in excess in diabetes only, we may still be content with the term "diabetic asthma."* We shall isolate the dyspnoea of continued exercise, it is produced by excess of lactic acid; and we shall place in a provisional and single group asthma associated with renal and cardiac disease, and may have to add to these a form of dyspnoea encountered at high altitudes. Taking the last group we may, sooner or later, come to regard the asthma in each case as the same, and due to a single error in metabolism, renal or otherwise; we may, on the other hand, be forced to place them separately; but at present, while asthmas which occur under these three sets of circumstances (renal, cardiac, high altitude) have much in common, they should be retained in a single class.

My reason for suggesting a classification with this basis is not a purely theoretical one; it has considerable practical consequences. For one and the same patient may, as we have seen, have dyspnoea from combined causes, and these causes may vary in intensity, relative to each other, in different patients and in the same patient from time to time; it is important that the cause or causes of hurried breathing should be recognized in a given case, for each has its own significance, and each calls for separate and appropriate remedies.

PROGRESS OF THE PATIENTS.

The further history of these patients in whom relative acidosis is discovered is variable. The majority are the subjects of disease which speedily terminates life. In many there is progressive emaciation; the constant distress and want of sleep rapidly fatigue and weaken the body, and exhaustion is profound at the end. In others, signs of cardiac failure are more prominent; they become more and more waterlogged and cyanosed, breathing becomes still more difficult, and the heart dilates; death appears to be due to cardiac failure. In others death is ushered in by cerebral attacks, and the patients die comatose or hemiplegic. As Drs. Ryffel, Cotton, and Wolff have shown, the picture may become complicated in the terminal phases by a retention of urea and of lactic acid. Inflammatory exudates are often developed; they may be limited to a single pleura or may affect both cavities, the pericardium and the peritoneum. When such appear, the temperature often rises to normal or above it before death. Draining the pleura is often ineffective, reaccumulation is rapid.

In some few patients the progress towards recovery, especially in those in whom signs of cardiac stasis are few, takes place. The dyspnoea vanishes, or is present only on exertion. Permanent and complete recovery is not known; relapses are frequent, and sooner or later the disease is fatal.

I do not propose to deal with the subject of treatment at all fully; it is equally variable with the symptomatology; usually it combines the treatment of renal and cardiac disease. I shall deal with special points only. For some of the patients a good deal can be done by treatment. Rest alone may be attended by considerable improvement. The dietary, as Huchard has stated, is important. Many of these patients are aware beforehand that a milk and vegetable dietary is the most suitable for them. A relatively salt-free diet seems to cause improvement in some instances. In patients who have dropsy the outlook is always serious, but some respond to diuretics, and those

* But if they also occur in starvation, we should return to a chemical terminology, defining the asthma as one resulting from a given error of metabolism.

who have fibrillation of the auricles may be greatly benefited by digitalis. Drainage of the legs should be carried out only with the strictest precaution. The incisions are prone to suppurate. Oxygen, even though administered in concentrated form, usually has little influence upon the distress of breathing. When the distress is great morphine alone affords relief. I have seen considerable relief follow free venesection, the breathing becoming quieter and sleep being induced.

The clinical signs sometimes show improvement on the cardiac side alone, so that cyanosis vanishes, engorgement and dropsy disappear, while the breathing still remains hurried, and bodily exercise remains difficult or impossible, because non-volatile acids still retain the blood in a state of diminished alkalinity.

I have given large doses of carbonates to several patients in the hope of altering the blood reaction; a difficulty in rendering the urine alkaline is often experienced, and although improvement has been seen in some patients, I cannot as yet speak of the remedy very hopefully. In the main our remedies are at present palliative, and temporary alleviation is alone anticipated.

THE ORIGIN OF THE ACIDOSIS.

Patients who suffer from the acidosis I have described have prominent lesions in two chief organs, the heart and the kidneys. In none of our cases were either of these organs found healthy after death. We seek the cause of the acidosis in defects of the circulation or in defects of the kidneys. That it is not due to deficient circulation is, I think, evident, for the degree of acidosis is not parallel to the circulatory defect. Is it the result of defective elimination, or does it come from an altered metabolism secondary to renal disease? To these and similar questions no certain answer can be returned; though we suspect that, as in one renal patient urea may be retained, and as in another chlorides may be retained, in a third type there is an alteration in the relative elimination of acids and bases. But whether this is the case or not is for the future to decide.

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An Address

ON THE

CLINICAL AND BACTERIOLOGICAL ASPECTS OF LEPROSY.

DELIVERED AT THE ROYAL SOCIETY OF MEDICINE,

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LEPROSY.

On considering leprosy from a nosological point of view we are struck by its long incubation, its slow and chronic course, and the terrible mutilations and disfigurement it is capable of producing without appreciably shortening the life of its victims. Its general infectivity, or, more correctly, contagiousness, appears to vary somewhat in different climates, because, though nowadays the disease, estimated by the number of cases, is practically confined to tropical and subtropical countries, still it is a universal disease occurring from north to south all over the inhabited world. In other words, it appears to have spread or persisted under some conditions and died out under others, for we know that it was extremely prevalent in Europe in the Middle Ages.

THE PATHOLOGY OF LEPROSY.

Its Contagious Nature.

It is not possible, from a scientific point of view, to deny that leprosy is contagious, even though we may not quite exactly know in which way the disease is transmitted from one individual to another. Knowledge of its