An Address

ON

PULMONARY OEDEMA.

DELIVERED BEFORE THE BEXHILL MEDICAL SOCIETY.

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AFTER some introductory remarks, Dr. Wethered proceeded:

Pulmonary oedema is a subject which receives but scant notice in the textbooks and other volumes dealing with general medicine which crowd the bookshelves of our libraries and consulting rooms at the present day. It is, however, a condition constantly met with in clinical work, and, owing to the fact that under certain circumstances it may prove most serious in itself, and at other times resemble other grave maladies, the question of diagnosis is often a source of considerable anxiety and concern to the medical attendant.

I need scarcely remind you that oedema of the lungs is not a primary disease, but is secondary to some pre-existing malady. Pulmonary oedema is, as you know, very commonly seen in the *post-mortem* room. It does not necessarily follow that it occurred during life, as it may have developed during the lethal agony. You may, perhaps, remember Cohnheim's remark: The patients do not die because of the oedema, but the oedema develops because the patient is dying.

For clinical purposes pulmonary oedema may be divided into three classes—acute, subacute, and chronic (or a better term would be “persistent”).

ACUTE PULMONARY OEDEMA.

I do not intend to consider at length the pathology or mechanism of this condition, but would merely remark that acute oedema of the lungs may be an outcome of most of the more severe forms of both acute and passive congestion of the lungs, those being also secondary processes. Authorities differ as to the exact manner of its production; many, however, favouring the view that owing to obstruction of the pulmonary circulation due to cardiac failure or other causes, there is some interference with the proper circulation through the lymph channels, and exudation accordingly ensues.

Acute oedema of the lungs is especially prominent in death by asphyxia brought about by pneumonia and acute bronchitis. In croupous pneumonia we know that when only one pulmonary lobe is attacked, the prognosis in a large measure depends upon the condition of those parts of the lungs not affected by the pneumonic process. If the opposite lung becomes greatly congested, acute oedema may develop, and the case then becomes most grave. In my experience this complication is more likely to occur in elderly patients than in younger subjects, and also in those patients exhausted by previous disease. I think also it is more frequent in pneumonia complicating influenza than when the pneumonic fever is primary. In alcoholic subjects also oedema is likely to develop, whilst the degree of toxæmia also probably has an effect in inducing this condition.

As a rule, oedema affecting the lungs follows the laws of gravitation, and the physical signs are generally to be sought for over the bases of the lungs, at any rate in the early stages of the process, but in acute bronchitis the oedema, when it occurs, not infrequently has a peculiar localization. It tends to take place more especially in the middle and upper parts of the lungs, consequently on listening over those areas the characteristic sharp, crackling râles are heard.

The occurrence of acute oedema of the lungs is facilitated by a hydraemic condition of the blood, and it is therefore not infrequently the cause of death in inflammation of the kidney, especially in acute nephritis. It is especially liable to occur if cardiac disease coexists with the renal condition. An attack may then supervene even after the disappearance of other dropsies. Pulmonary oedema may also be the final stage in some of the disorders of the blood, such as pernicious anaemia, and is very occasionally seen also in cases of scurvy. The occur-

rence of acute pulmonary oedema in connexion with the diseases I have mentioned has received general acceptance, and we should always be on the look-out for its possible development in patients suffering from those affections, but there are other considerations under which it may occur producing rapidly fatal results, and some of these I now propose to consider. The first I will mention is cardiac disease. Subacute and chronic pulmonary oedema is of common occurrence in cases in which the circulation is carried on under difficulties. Acute oedema, however, is, I believe, rare. A few years back I was asked to see a gentleman aged 73, who had always lived in easy circumstances. His only complaint was indigestion. He was greatly troubled with flatulence after meals, and constipation was a source of constant annoyance to him. These symptoms had persisted for many years. He was able to walk a considerable distance at a moderate pace, but if he hurried he experienced slight dyspnoea. There was no history of pain across the chest after exertion. I was surprised to find his pulse-rate 140 per minute, but there was no irregularity of the beats. The arteries were in a fairly good condition considering his age, and the tension was slightly increased. On examining his heart I found it slightly dilated, the sounds being clear. There was no albumen in the urine. I saw him at intervals for some months and the dyspeptic symptoms became less marked. One day I was called to see him as he thought he had taken cold. I found him complaining of cough and shortness of breath. A few rhonchi were heard over both lungs, anteriorly and posteriorly. The heart was beating very rapidly (160 per minute) but the rhythm was perfectly regular. On the next day numerous râles were heard over both lungs, and there was impairment of resonance over both bases. The pulse-rate was again 160, and the second sound of the heart could scarcely be heard. Dyspnoea was extreme, and he was expectorating a large amount of thin watery fluid. Death occurred two days subsequently in spite of all the means that were tried to relieve the condition. This was a typical case of acute pulmonary oedema occurring in conjunction with a weakened heart, and indicates the possibility of its occurrence in a patient who had not previously exhibited the usual symptoms of cardiac failure.

It is well known that rapid unforeseen death may take place after the performance of paracentesis thoracis. Acute oedema of the lungs is supposed by some authors to be the cause of death in some of these cases. Personally I have never met with a case which ended in this way. I shall have to refer later to persistent oedema of the lungs after tapping the chest, but when acute oedema does occur under these circumstances it is stated to be one-sided only (the side that was operated on) and has been attributed to the rapid expansion of the lung which has been compressed for some time. It is possible that the blood vessels become suddenly distended with blood, and exudation results. Most authors state that oedema thus developed is attended with much less grave results than would have been expected considering the very copious expectoration of clear fluid which occurs.

Another cause of acute pulmonary oedema is sudden mechanical obstruction to the blood vessels of the lung. This accident is most likely to happen in the form of thrombosis of the main pulmonary veins, or in embolism or thrombosis occurring in many vessels of the lung, generally in gouty subjects.

Other conditions which may be the cause of acute pulmonary oedema are certain of the specific fevers accompanied with hyperpyrexia. Perhaps the oedema occasionally occurring in the course of diphtheria may come under this head, also that which is met with as a rare complication of whooping-cough. Such oedema has been ascribed to a toxæmia, and I should think that this is very probably a correct explanation. Certain poisons may cause death by pulmonary oedema, mainly by producing rapid cardiac failure.

There is one other condition under which acute pulmonary oedema may be produced, and that is after the administration of an anaesthetic. It most frequently occurs after ether has been employed. I recollect one case which greatly impressed me. It was that of a gentleman, aged 54, who sought advice on account of profound anaemia. Subsequently an obscure swelling was found in the right flank, and an operation was performed in order to ascertain the nature of the tumour, and

whether it might be possible to remove it. It turned out to be malignant disease of the colon, the removal of which could not be undertaken. Twenty-four hours after the operation the patient complained of severe dyspnoea and distressing cough. On examination there was found impairment of resonance over the base of the right lung, and subsequently over the left, with bronchial breathing and numerous high-pitched râles. The expectoration was copious and blood-stained. The condition was ascribed to "ether-pneumonia." Death occurred on the fourth day, and at the autopsy the lungs were found in a highly oedematous condition, but there was no pneumonic consolidation. Possibly the anaemic condition in this case predisposed to the occurrence of oedema.

The symptoms of acute pulmonary oedema may be judged from the cases I have mentioned above, dyspnoea and straining cough being the most constant. These symptoms, however, may be caused by the primary conditions, and are not necessarily due to oedema, but a rather sudden increase of the dyspnoea, with increasing cyanosis developing in the course of the diseases I have mentioned as being more or less frequently complicated by acute pulmonary oedema, should give rise to a suspicion of the onset of this serious condition. The only certain diagnostic phenomenon of acute pulmonary oedema is a copious, thin, watery expectoration, sometimes, when the secretion is very abundant, tinged or streaked with blood. This symptom, however, is not a constant one, since the patient may die before expectoration occurs. Extreme exhaustion may be produced by the primary malady, and consequently the patient has not strength enough to cough up the fluid, and hence death occurs by suffocation.

A state of orthopnoea generally exists, and the respiratory movements are mainly thoracic, the thorax moving as a whole, the lower portion receding with inspiration. On percussion the note will probably be found to be hyperresonant in front, but posteriorly over the bases it will be more or less impaired. On auscultation a few râles may be heard over the front of the chest, but posteriorly, extending from the base downwards, the breath sounds are feeble, or perhaps annulled, and fine bubbling râles are heard mainly with inspiration. The râles are similar to those heard along the anterior margins of the lungs in emphysema, or the sounds that are heard over the pulmonary bases when patients who are not accustomed to breathe properly are directed to take a deep inspiration. When the chest first expands under these circumstances a peculiar fine creaking is heard, which disappears after the first two or three inspirations. The râles produced by oedema also somewhat resemble the crepitations heard in cases of pneumonia, hence confusion in diagnosis may arise. For instance, in cases of pneumonia where the base of one lung is involved, the development of oedema in the opposite lung may afford a suspicion that the pneumonic process has spread to the other lung.

From these remarks it will be seen that not infrequently the diagnosis of acute pulmonary oedema is one of surmise only, but the subsequent course of events will prove the true state of affairs.

I need only say a few words in reference to the morbid anatomy of acute pulmonary oedema. The lungs are large and heavy; they are indented by the ribs and pit on pressure. On section, an abundant frothy fluid exudes, which is sometimes blood-stained. All these characters are usually most marked at the bases and may only be apparent there unless otherwise locally determined, as I mentioned when speaking of the oedema accompanying acute bronchitis. One point must not be forgotten, and that is, that it is very usual to find the pleura on one or both sides containing a large amount of serum. Except at the extreme bases, the lungs are generally more or less crepitant and portions of the organs when placed in water float. An exception to this may occur when, owing to old-standing mischief, due probably to chronic cardiac disease, the lungs have become thoroughly permeated with fluid. On microscopic examination, some epithelial debris may be observed in the alveoli, otherwise the texture of the lungs is unaltered if oedema only is present.

With regard to prognosis, acute oedema of the lungs is always of grave significance. It is one of the later phenomena in nephritis, and in cases of failing heart, whether from valvular disease (particularly of the mitral

valve) or from affections of the myocardium. The super-vention of pulmonary oedema in acute bronchitis or in croupous pneumonia is of almost certain fatal augury.

The treatment of acute pulmonary oedema will naturally vary with the primary cause. The most valuable remedies consist in cardiac stimulants—strychnine, best administered hypodermically, commencing with $\frac{1}{15}$ gr., $\frac{3}{16}$ gr. or $\frac{1}{8}$ gr. being afterwards given according to the severity of the case; digitalis and strophanthus in appropriate doses, and I have often found that the two drugs may with advantage be given in combination. I have also obtained encouraging results by the hypodermic injection of these drugs together with strychnine. Brandy is also a useful ally. Squill may also be useful, both as a cardiac stimulant and an expectorant. I always employ oxygen gas, given at regulated intervals through a mouthpiece, direct from the cylinder without the intervention of a balloon, or the gas may be given continuously. It then serves not only to keep the blood aerated, but also aids in procuring sleep and the much-needed rest. Dry-cupping to the back of the chest may also be tried. With regard to venesection, great caution is necessary in its employment, and if the presence of oedema is certain I should hesitate to adopt this procedure unless as a last resource. Some observers, however, have recorded success in cases which appeared most desperate. There is no doubt that the prejudice against bleeding has been carried too far, and that this therapeutic measure is a most useful one in relieving a dilated right heart. I need scarcely say, however, that it should not be resorted to without full consideration of all the elements in each individual case.

SUBACUTE OEDEMA OF THE LUNGS.

This condition I believe to be more common than is generally supposed. It is often one-sided; in other cases both bases may be affected. With regard to the latter class I need say but little. Such a state of affairs is perfectly familiar to you as likely to occur in patients who are the subjects of emphysema, when any increase in the bronchial condition may speedily give rise to a certain amount of oedema which gradually subsides when cardiac tonics and stimulant expectorants are given. Patients suffering from myocardial degeneration, or insufficiently compensated mitral valve disease, especially mitral stenosis, are also liable to present similar phenomena, especially after unusual exertion to which the damaged heart has been unable to respond. Treatment consists in giving digitalis and strychnine. A course of "blue pill" will also be found efficacious. I wish, however, to refer more particularly to subacute oedema affecting one pulmonary base only, as errors in diagnosis may easily be made. The physical signs in such cases consist of small or medium-sized bubbling râles at one base (most frequently the left), which exist for a certain length of time and then disappear. There may or may not be some impairment of resonance over the same area, together with weakening of the breath sounds or prolongation of the expiratory sound.

One condition under which such a collection of signs is met with is in the later stages of an attack of influenza. After the acute symptoms have subsided a racking, paroxysmal cough develops, and on examining the chest one or more of the physical signs I have just mentioned will be found. The cough is often most distressing, and frequently proves very intractable to treatment. The patient suffering from the post-influenza debility is still further shaken by the violence of the cough, and hence convalescence is considerably retarded. The friends are naturally apprehensive lest some more serious malady is threatened. The medical attendant, conscious that influenza may prepare the tissues of the lung to be a favourable pabulum for the tubercle bacillus, and thus afford the "second etiological factor" necessary for the development of pulmonary tuberculosis, also feels no little anxiety until the disappearance of the cough and full return to health on the part of the patient reveals what was the true nature of the cough. After making trial of numerous drugs to relieve the cough, I have obtained the most satisfactory results from the use of hydrochloride of heroin given in tabloid form in doses of $\frac{1}{15}$ gr., the tabloid to be sucked and not simply swallowed. Counter-irritation to the base of the lung also gives relief.

Some physicians would perhaps classify such cases as undeveloped influenzal pneumonia. The general con-

ditions, however, are, as a rule, scarcely severe enough, in my opinion, to warrant such a diagnosis.

Another class of case is met with which closely resembles those I have described as dependent upon influenza, and indeed the differential diagnosis may be disputed. I have seen this class of case most frequently in London, and generally in the months of August and September, and the outbreak appears to be epidemic in character. The illness commences with a sore throat, gradually increasing in severity. There is moderate pyrexia, the temperature rising to 101° or 102° . Laryngitis follows on the pharyngitis, and then cough becomes a prominent symptom. This is paroxysmal and tearing in character, often producing considerable though temporary pain in the head. When the sore throat has subsided the cough persists. There may subsequently be a daily rise of temperature to from 99.5° to 100° F. Expectoration is scanty at first, subsequently becoming more abundant and muco-purulent in character. General malaise is more or less marked. On examination, fine bubbling râles are heard over one pulmonary base, rarely over both bases. The cough often becomes very trying, being almost persistent, and disturbing sleep at night. The debility accompanying the attack is marked. Headache, except after cough, is not a prominent symptom, and aching of the limbs is not, as a rule, complained of. I believe the attack to be caused by some poison being inhaled into the system, and I think it is very probable that, as has been suggested, it is brought about by the dust which collects during the hot months of the year on the wooden pavements. Dry horse dung, and the dust from the house refuse as it is being emptied into the "sanitary dust-carts" and so profusely distributed, doubtless add to the malignant nature of the ordinary road scrapings. This mixture is scattered by the traffic, and if there is any wind must be freely inhaled by persons passing along the streets. It is, I believe, further intensified by the plan adopted in some parts of London of only watering the sides of the roads during the daytime, leaving the central portions dry. Whether this is from motives of economy in labour and water, or whether it is intended to help the horses by not rendering the roads greasy by means of the water, it is difficult to say. At any rate, I should urge that this etiology of the condition I have described is worthy of serious consideration.

The treatment of this affection is unsatisfactory, and I have frequently seen the symptoms persist for two months or longer, the general health of the patient thereby suffering considerably, and rendering the tissues, especially those of the lungs, vulnerable to bacterial attacks. Iodide of potassium, carbonate of ammonia, tincture of nux vomica, and syrup of tolu may be given in a mixture, and hydrochloride of heroin frequently relieves the cough. Change of air and rest from the ordinary duties of life will probably constitute the best prescription that can be ordered.

Under the heading of subacute oedema of the lung, I would place the oedema that occasionally occurs after the withdrawal of a pleuritic effusion, excluding those acute cases to which I have already referred. Usually the physical signs make their appearance at the posterior lower part of the thorax on the side on which the effusion took place. Impaired resonance, weak breath sounds and voice sounds and bubbling râles. In a certain number of these cases the oedematous condition is situated in the pleura and gradually disappears under the influence of fresh air, good food, and such drugs as preparations of strychnine and iron. Sometimes, however, there is another distribution of the oedema, as in a case brought before the Clinical Society of London by Dr. J. Calvert, in which for four months after paracentesis numerous râles were heard over the upper part of the affected side, and the patient expectorated large quantities of serous fluid. Dr. Calvert made the suggestion that the oedema was brought about by the existence of adhesions which, as the lung expanded, compressed the pulmonary veins coming from the upper lobe.

Another cause of subacute oedema of the lung to which I would draw attention is the existence of adhesions the result of previous pleurisy. When a patient with this condition acquires a chill (using the ordinary term without inquiring minutely into what is really implied by it), temporary oedema will often be found at the site of the adhesions, but whether the pleura or the lung is the site of the oedema it is difficult to say.

The diagnosis of such cases presents some difficulties. They may be confused with several other conditions. One which may be mentioned is what is usually termed "unresolved pneumonia." In a few cases of pneumonia, instead of the physical signs all disappearing in due course as the pneumonic process subsides, impaired resonance and râles remain for a certain length of time at the affected base. This is asserted by some physicians to be caused by imperfect resolution. I am inclined to believe that the râles are produced by oedema of the lung or pleura continuing after the acute condition has passed off. Here I may add that undoubtedly many cases of so-called unresolved pneumonia in children are really cases of empyema, the suppurative condition following on the pneumonic change and having the same microbic origin—the pneumococcus. Occasionally a similar course of events is observed in the adult.

After an attack of acute or subacute bronchitis the same physical signs may present themselves. The history of the case is, of course, of importance in interpreting these signs.

Other difficulties in diagnosis consist in distinguishing the above-mentioned conditions from those occurring in the class I am about to consider, namely:

CHRONIC OR PERSISTENT OEDEMA.

I would rather have entitled this part of my subject: "Râles heard at the base of one lung," for such is the only physical sign usually manifested, although occasionally the percussion note may be slightly impaired and the expiratory sound somewhat modified.

The causes of such râles are varied and may be a source of much perplexity. In old people with a feeble heart or emphysematous lungs, persistent as well as subacute pulmonary oedema is common; usually the râles are constant at the base of one lung, but are occasionally heard over both pulmonary bases. Sometimes, however, both bases are permanently affected in this manner. Such an event is especially liable to occur when the individual is the subject of chronic renal disease or of glycosuria. If either of these two conditions are present the prognosis must be a guarded one. Death due to the kidneys is the most common form of unforeseen sudden death. The fatal termination may then be brought about in various ways; but if the circulation is feeble, as demonstrated by persistent oedema of the base of one or both lungs, there may be serious danger ahead at any time. Diabetes is perhaps the disease of all others which runs the longest course without its subject being aware of it, whatever may be the quantity of sugar voided in twenty-four hours. If sugar is found in the urine, even in small quantities, and at whatever age, I always regard the life of the patient as a precarious one. Some of the subjects thus affected do certainly live to a good old age, but sudden death through the heart is common, and if râles are persistently heard over one of the pulmonary bases cardiac weakness is indicated, which naturally adds to the gravity of the prognosis. These patients are sitting, as it were, on the brink of a volcano, which may remain quiescent for a long period, but an eruption may at any time take place with the most serious and immediate consequences.

Anaemic conditions, combined with râles at one base, must also be regarded as suspicious. The blood in anaemic states is hydraemic, and consequently oedema is readily produced. The class of case I have more particularly in my mind is that in which pernicious anaemia or leucocythaemia exists and has not been recognized. If profound anaemia is accompanied with râles at the base of one lung the blood should always be examined with reference to the numbers and forms of the red corpuscles, the percentage of haemoglobin, and a differential estimation of the various forms of leucocytes.

Another important class of case in which sometimes râles at the base of one lung form the only physical sign is that in which there is some pressure on the bronchus and vessels of the lung, generally on the left side. Aneurysm of the aorta only signifies its presence, at any rate in the early stages, by signs of pressure. The diagnosis may be easy, as when there is a constant gnawing pain, difficulty in swallowing, a characteristic cough, and a visible pulsating swelling. But if the aneurysm is situated on the descending arch or on the descending thoracic aorta, the physical signs and symptoms may differ very widely, and owing to the fact that

no important structures are pressed upon the diagnosis may be most difficult. Especially if pain be absent, the true state of affairs may easily be overlooked. In some of such cases, the only symptoms are cough (which may not have the characteristic ring) and increasing dyspnoea; on auscultation some râles may be heard at the left pulmonary base. Such symptoms and physical signs may easily be ascribed to other causes. The lungs may be emphysematous, still further complicating the difficulties of diagnosis. When examining patients exhibiting these symptoms and signs, the aortic second sound should be carefully noted. Then, if an aneurysm be present, the characteristic ringing second sound is heard accompanied by a distinct jog to the ear, when a wooden stethoscope is employed. The presence of this phenomenon should always give rise to the probability of aneurysm being present. In the cases to which I am now alluding the diagnosis may be corroborated by the presence of tracheal tugging, or the conduction of tracheal sounds to the margin of the manubrium sterni. If the aneurysm is situated on the descending thoracic aorta the diagnosis will be still more difficult. Indeed, if pain be absent, as not infrequently occurs, the exact morbid condition may remain entirely unrecognized. Then we only have the cough, dyspepsia, and râles at the base of the left lung to guide us, and these phenomena may, as we know, be due to many causes. The *x* rays may then lend valuable aid in diagnosis; the shadow yielded by an aneurysm being plainly visible, although errors are easily made, unless the shadow cast by a healthy aorta is fully known.

I have laid stress on the above points in order further to accentuate my contention that these râles may be indicative of grave organic disease, and may be the only physical sign to warn us that some obscure lesion is present.

I will here relate a case which shows the difficulty which arises in cases of this kind.

A man, aged 56, had been in the Middlesex Hospital, having been admitted for hæmoptysis, but at that time there were no definite physical signs. He subsequently attended under my care as an out-patient. I found a faint diastolic murmur over the aortic area, and numerous râles just above the angle of the scapula on the left side. He complained of no pain whatsoever, and was thought to be suffering from aortic regurgitation. Some time afterwards he was again admitted into the wards with profuse hæmoptysis, and died in about three hours. At the autopsy an aneurysm of the descending thoracic aorta was found, which had ruptured into the lung at a spot exactly corresponding to that over which I had heard the râles.

This case is a striking instance of the uncertainty which attends the occurrence of this disease, and the importance that may be attached in certain cases to the appearance of these râles.

Closely allied to aneurysm is the existence of other tumours in the chest indicating their existence only by pressure on a bronchus or vessels at the root of the lung. Carcinoma affecting a lung usually commences at the root, and may give rise to the physical sign I have been considering.

I may, perhaps, be here allowed to interpolate the warning that dyspnoea out of all proportion to the physical signs points to the possibility of carcinoma of the lung; the other indications, especially the wasting, only becoming marked at a later period of the case.

In children, also, enlarged glands may give rise to similar manifestations. In such instances, paroxysmal attacks of dyspnoea, closely resembling asthmatic attacks, may be observed.

The difficulties of diagnosis in such cases is obvious. As I have already indicated, râles at the base of one lung are present in many varying conditions. I will now briefly refer to one or two others in which they cannot be ascribed to persistent oedema of the lungs, but these diseases may easily be mistaken for some of those I have already pointed out. Râles at the base of one lung, not necessarily the left, may be the first indication of early basic pulmonary tuberculosis. I have seen several instances of patients attending in the out-patient department of Brompton Hospital complaining of cough, expectoration, and loss of flesh, the only physical sign being the existence of such râles, the subsequent course of the illness indicating its tuberculous nature. The examination of the sputum is, under such circumstances, of the greatest value, and I may insist on the importance of an examination for

tubercle bacilli in all cases where the diagnosis is obscure. Pulmonary tuberculosis commencing at the base instead of at the apex is uncommon, but this initial aide must not be forgotten.

Another lesion that may give rise to similar physical signs is bronchiectasis. The signs in this disease are frequently obscure. Râles may be present over the affected area of the lung, and if this happens to be the lower lobe we again have the sounds to which I am referring. In many cases there is no obvious impairment of the percussion note or alteration of the breath sounds. The râles, however, have a peculiar character, being sharply crackling and high-pitched. By some physicians they are known as "bronchiectatic râles." The odour of the breath, especially after coughing, and the peculiarities of the sputum may corroborate the diagnosis. The condition may, however, be confounded with pulmonary abscess; but in the latter disease the sputum is not so copious, and the physical signs are often very slight. Abscess of the lung affecting the base is, I believe, not common, but in other areas the differential diagnosis between gangrene of the lung, bronchiectasis, and localized empyema is often very difficult, but scarcely enters into the range of my subject to-day. I would only add that in bronchiectasis and abscess of the lung, the former especially, treatment by means of the creosote bath has given excellent results.

I do not suggest that I have exhausted all the causes which may give rise to temporary oedema; collapse of the lung may, for instance, give rise to temporary oedema, but is difficult to distinguish from other conditions. I trust, however, that I have said sufficient to show that the greatest care is necessary in arriving at a conclusion when signs of persistent oedema are found to exist.

Remarks

ON

ACUTE NEPHRITIS (MORBUS BRIGHTII),

WITH SPECIAL REFERENCE TO TREATMENT.*

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ALTHOUGH anasarca and ascites were well known to the ancients, and are referred to in the writings of Aretaeus and Celsus amongst others, and much later Contugno and Cruikshanks in the eighteenth century noticed that dropsy is often attended with the presence of serum in the urine, yet it was left to our renowned countryman, Richard Bright, to discover and establish the fact that many cases of dropsy were dependent on well-marked lesions of the kidneys, and that the urine in such cases was albuminous. His *Reports of Medical Cases*, published in 1827, containing a lucid description of 23 renal cases, with the *post-mortem* reports in some instances, is a lasting monument to his acumen and skill as a physician. Following Bright came many workers, notably Osborne, James Gregory, Christison, Barlow, Rees, and Wilkes in this country, and Rayer, Professor Frerichs, Jaccoud, and Bartels on the Continent. These men were divided into two schools, one led by Bright and supported by Rayer, which contended that the primary disease was situated in the kidney; and the other led by Graves, who in 1831 propounded that the condition of the blood was the primary disease, and the condition of the kidney which resulted from the irritation of the albumen passing through it was secondary. And there was truth in both of these views, as acute and chronic renal disease had not yet been differentiated, and Richard Bright himself, under his third heading, described "contracted kidney." It was Rayer in 1840 (*Traité des Maladies des Reins*) who first positively recognized the changes in the kidneys to be inflammatory, and stated that they could be acute or chronic. This view was proved conclusively by the histological investigations of Reinhardt and Frerichs in 1850 and 1851. Anticipating both these writers, Dr. George Johnson pub-

* A paper read before the Liverpool Medical Institution, February 15th, 1906.