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A Clinical Lecture

ON

OEDEMA OF THE LUNGS AND ITS IMPORTANCE AS A MODE OF DEATH.

Delivered at St. George's Hospital,

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THE search for specific remedies for diseases has not up to the present yielded any considerable harvest. It is true that we seem to be on the verge of a new epoch. Now that *materies morbi*, which to our fathers was little more than an abstract conception, has begun to take particulate form, and chemical analysis is diving into the secret of its mode of action on the body, we may reasonably hope that experimental science will one day surmount the further step of discovering the means of counteracting it. That step, however, is a long one, and at present we must reconcile ourselves to the fact that there are few remedies known to us to which we can even plausibly attribute the property of destroying or neutralizing the physical basis of any disease inside the human body.

We physicians, however, cannot therefore sit down idly and await further discovery. Our business is with the present. Disease, with its train of suffering and death, is in the field. If we cannot always destroy the enemy, we can at least place some obstacles in its path; strategy is open to us, though we may not have the force for direct attack; and by well-directed therapeutic strategy death may be avoided as well as suffering. In fighting disease delay is often equivalent to victory, and *cunctando restituit rem* may be written as well of therapeutic as of Fabian campaigns.

The tendency of acute diseases in particular to run a course definitely limited in point of time is not a newly-observed fact, though modern bacteriology has confirmed and partly explained it. If the patient survive the allotted time the gravest danger to life is past. He may, indeed, be left crippled, he may have sustained during conflict with the disease an organic lesion which will eventually cut short his life; but the acute illness is over. If no permanent organic lesion is left behind his recovery is complete. Such is the normal course of the greater number of acute diseases, without reference to treatment. In some few we have already the power of cutting the attack short; in most we can only endeavour to limit its effects on important organs, and to stave off a fatal issue until the time is passed at which the malady possessed the power to cause it.

Successfully to resist an enemy one must know something of his resources and methods. If we are to succeed in averting death from disease we need, above all things, to know how in each case the fatal result may be brought about. It is to this subject, or at least to a part of it, that I wish to direct your attention to-day. I do so the more particularly as it is a subject to which, I venture to think, adequate attention is seldom given. We are too apt to content ourselves with generalities. It is so easy to say that a patient died of

scarlatina, or cerebral haemorrhage, or granular kidney; so much more difficult to define how the malady actually brought life to an end. But this is often the really important matter; the malady itself we may not have the means of removing; its fatal effects we may perhaps find ourselves able to avert if we know precisely what they are.

Death means the permanent arrest of the heart and respiration, or practically of either, for neither can go on long without the other. It may be brought about by destruction or by temporary abeyance of function of any one of the active elements which are necessary to the cardiac or respiratory movements, from the nerve centres in the medulla oblongata to the haemoglobin in the red corpuscles, and it can occur in no other way. Older authors used to classify deaths into deaths occurring from the heart, deaths occurring from the lungs, and deaths occurring from the brain. This triple classification was unsound and misleading. There is no death from the brain except through the agency of the nerves and muscles of the heart, the thorax or the arterial channels; there is no death at all as long as the heart and respiratory muscles are still in action.

The mechanism of death, you will find, is in the great majority of cases neither obvious nor simple. When, indeed, a ventricle which is the seat of advanced fatty degeneration has stopped contracting, or a large aneurysm ruptured, or a mediastinal growth occluded the trachea, or an intracranial effusion been present in sufficient amount seriously to compress the medulla oblongata, it needs no profound physiological knowledge to tell us in what way the condition of things had become incompatible with the further maintenance of life. But such simple cases are the exception, and from the examples I have quoted you will be correct in judging that the most obvious modes of death are, generally speaking, those that least admit of remedy.

Some cases of poisoning, again, show us the mechanism of death in a simple form. We have poisons that kill, like morphine, by paralysing the respiratory centre; like curare, by paralysing the motor terminals of the respiratory nerves, like carbon monoxide, by paralysing (if I may so call it) the haemoglobin; like chloral and prussic acid, by bringing both heart and respiration to a standstill. To these modes of death we oppose, and with some measure of success, equally simple and direct remedies. We have no means of destroying or neutralizing (in the chemical sense) any of these poisons, once they have been absorbed into the circulation; but in the less severe cases, by means of atropine and artificial respiration, for example, we may succeed in keeping going the respiratory function, or by means of digitalin that of the heart, until such time as the excretory glands have been able to eliminate the absorbed poison from the fluids of the body, and the danger to life is past.

In the greater number of cases of disease the problem is far less simple. We know that in microbic diseases definite poisonous substances are thrown into the circulation; we know that in some forms of organic disease equally definite poisons are retained in it, and some at least of these poisons seem to possess a directly depressant action on the cardiac and bulbar nerve tissue; but in clinical experience it is exceptional to witness a death directly and solely due to such intoxication of the cardiac or respiratory nerve apparatus or the muscles which they actuate. In by far the greater number of instances the immediate cause of death is to be traced in some more tangible lesion of vital organs; but I must warn you that even in diseases which essentially involve

lesions of the heart or respiratory organs the proximate cause of death is seldom that which a *prima-facie* knowledge of the disease would suggest. In illustrating this I shall have particularly to ask your attention to a lesion the clinical significance of which is often overlooked, and which has hardly received the measure of attention in medical literature that its real importance seems to me to claim. The lesion I mean is oedema of the lungs.

In cases of disease of the heart valves, to take an example, it is exceptional to find death occurring primarily from stoppage of the heart. The damaged heart, in fact, seldom stops as long as the respiration is free. I have often invited your particular attention in the wards to the phenomena of mitral stenosis in its successive stages. The narrowing of the left auriculo-ventricular orifice, if duly compensated by adequate hypertrophy of the right ventricle, may exist, as we almost daily see, for many years, not, indeed, without causing troublesome symptoms, but at any rate without immediately threatening a fatal issue. At length the day comes when compensation fails. The right ventricle gives way under the strain. Perhaps the stenosis has increased beyond the capacity of the ventricle to compensate; perhaps bronchitis has aggravated the obstruction in the pulmonary circulation; perhaps the nutrition of the thickened ventricular wall has begun to fail. The ventricle dilates; the right auriculo-ventricular orifice becomes stretched; the tricuspid valves are therefore incompetent; and the scene is changed. The systemic and portal veins become engorged, oedematous effusion appears in the lower extremities and mounts upwards, the liver swells, and the obstructed portal circulation unloads itself into the peritoneal cavity. The patient is now quite disabled; his legs waterlogged, his abdomen distended, his heart struggling to overcome the deficiency of its valves, his breathing oppressed, and his blood insufficiently aerated. But in all this disablement, serious as it is, there is as yet little of a *fatal* element. Oedema of the extremities does not kill, unless cellulitis supervene. Digestive disturbances and impaired nutrition kill but slowly. Ascites, it is true, may hinder the respiration by preventing the descent of the diaphragm, but ascites may be relieved, in some cases many times in succession, by paracentesis. In such a condition, severely ill, but not immediately threatened with death, the patient may linger weeks or months before fatal lesions appear; he may even improve under treatment and partly recover. Danger is chiefly to be looked for neither in the extremities nor in the abdomen, nor even in the heart, the original source of the trouble, but in the lungs and the pleural cavities. In these regions several untoward events may happen. The systemic dropsy may extend to a pleural cavity; though this, indeed, may be relieved, like ascites, by paracentesis. Infarction of the lung may take place by means of emboli from the dilated right ventricle; pneumonia may intervene; but by far the commonest danger to be apprehended is the extension of the dropsy to the lung itself.

You may have often seen me, when brought for the first time to a severe case of heart valve disease in the wards, without waiting for a detailed examination of the heart itself, proceed at once to make the patient sit forward, and begin by investigating the state of the bases of the lungs. It is there that the most significant signs of immediate danger are to be found; and oedema is the condition most to be suspected. Cardiac oedema is a passive oedema, and usually therefore appears first in the most dependent parts. In its early stages it interferes little with the resonance of the chest, and it makes no change in the vocal fremitus or vocal resonance; but on deep inspiration the ear will detect a number of clear, fine, "fluid" crackles, of uniform size. The prompt recognition of the nature and significance of these crackles is highly important, and with a little practice it is not difficult. The rhonchus, and sometimes the *râles*, of bronchial catarrh may be present with them, for the process may be actually affecting the minute bronchi as well as the lungs, or there may have been a pre-existing bronchitis. It is essential, therefore, to learn not to confuse the two sets of signs, for the oedema is of far graver import than the bronchial catarrh. The rhonchus can cause no difficulty; but practice alone will enable you certainly to distinguish between the "mucous" *râle* of bronchial secretion, and the clearer, sharper, more "fluid" sound of oedematous effusion into the lung itself.

As the process progresses, the crackles are heard extending from below upwards, and larger, but still sharp and "fluid" *râles* become mixed with them in increasing proportion. If the oedema is severe the percussion note becomes impaired; if the lungs in any part become completely waterlogged dull-

ness may be complete, and breath sounds over the region entirely abolished; but the persistence of the voice sounds and vocal fremitus enable the condition to be distinguished without difficulty from one of pleural effusion.

The subjective symptoms of pulmonary oedema are few, and not distinctive. Distress of breathing, needless to say, is increased by it; cough is often increased, and becomes more hacking; but expectoration of the oedematous fluid occurs only when the oedema is extreme. In slighter grades of oedema there is no *vis a tergo* to expel the fluid, as there is in the case of bronchial secretion, the fluid being in the air sacs themselves. There is little, therefore, in the symptoms to draw particular attention to the onset of oedema; it has to be watched for by regular and systematic examination of the back of the chest.

Oedema of the lungs, though for the time and even more than once it may be checked by treatment, usually proves sooner or later the last scene of the drama. When treatment finally fails to control it, the *râles* become more and more abundant and large; the respiration-rate goes on mounting higher and higher as long as the reserve forces of the respiratory apparatus hold out against the increasing demands upon them; cyanosis becomes more and more marked; until at last the reserve of respiratory force begins to fail, and the respiratory movements grow slower and shallower, till in the end they stop altogether. The end, however, is not always purely by failure of respiratory movements; the obstruction to the passage of blood in the lungs adds to the strain already suffered by the right ventricle, and the damaged heart may stop somewhat suddenly as things are rapidly nearing the fatal end, even before the respiratory power is quite exhausted. In either case, nevertheless, necropsy plainly reveals the waterlogged state of the lung tissue which is the real method of death.

I have spoken here for the sake of clearness of mitral stenosis; but the case of aortic regurgitation is not very different. In aortic regurgitation the compensation is made by a marked hypertrophy with dilatation of the left ventricle. As long as the hypertrophied ventricular wall remains sound no serious results are likely to occur. The downward course is generally started by such additional dilatation or partial degeneration of the left ventricle as renders the mitral valves insufficient and throws part of the work of compensation on the right ventricle, which in the actual state of the heart at the time has preserved little power of hypertrophy, and ere long undergoes dilatation in its turn, causing the tricuspid valves to be incompetent and systemic dropsy to set in. What I have just said of the final stage of mitral stenosis may be said with little variation of almost any common form of heart-valve disease; in all these cases alike oedema of the lung is the danger chiefly to be dreaded, the condition most often calling for treatment in the last resort.

In acute croupous pneumonia, to take a second illustration, we might think we discerned an obvious method of death in the inflammatory consolidation that is its most constant lesion; but I have frequently pointed out to you in the wards, on the one hand, how large an extent of lung tissue we may find involved in the inflammation without its causing or even seriously threatening a fatal termination, and in the *post-mortem* room, on the other hand, how small a portion may be found inflamed even in a rapidly fatal case. "A patient," says a recent writer, "may die of pneumonia, and yet the area of consolidation be not larger than a small orange." The average extent of lung found consolidated after death is indeed much less than that which is found destroyed or rendered useless in many cases of pleural effusion or tuberculous disease, in which, nevertheless, life is preserved for a far longer period than would cover the limited duration of a pneumonic attack. Consolidation alone is obviously not always the precise cause of death. But, as I have also taken occasion to point out to you on the *post-mortem* table, the unconsolidated portions of the lung in a fatal case of pneumonia are seldom in a state of health. Large tracts of them, if not the whole, are usually the seat of a morbid condition nearly as detrimental to their utility as inflammation. They appear after death fully distended, with the surface stretched smooth, and their cavities are found on section to be more or less completely filled, to the exclusion of air, with a clear albuminous fluid exuded from the blood. They are, in a word, affected by a severe oedematous process.

Now we cannot regard this oedema as a mere terminal or *post-mortem* change, for two plain reasons: that the distension

of the lung tissue is usually too great to be the result of such passive oozing, and that the physical signs of oedema have often been heard during life; heard, I mean, not merely *in articulo mortis*, but for hours or days before death in a fatal case, and occasionally for hours or days in severe cases that ultimately end in recovery. Nor can the theory be deemed tenable which represents the oedema as but a secondary and mechanical phenomenon, the result of engorgement of the pulmonary veins from failure of the left ventricle of the heart owing to the direct action of pneumonic toxins. This theory, I believe, reverses cause and effect. Neither in the phenomena of a mild case of pneumonia, nor in those of the early stage of a fatal case, do we see anything to suggest, as we do in cases of typhoid, diphtheria, and influenza, that the poison of the disease exerts a marked and direct depressing action on the heart. On the contrary, the characteristic pulse of pneumonia is full and bounding, the pulse-rate is seldom excessive, and the face usually wears a bright flush.

Nor is oedema of the lung by any means so easy to produce by mechanical means as this explanation assumes. Obstructive oedema, even in the area of the systemic circulation, requires either one of two conditions—a fairly complete obstruction, or a sufficient duration of obstruction to allow of changes taking place in the walls of the distended vessels. And mechanical oedema of the lungs is, if anything, less easy to produce than systemic oedema. Let me remind you again of the phenomena of mitral stenosis. We saw that in a well-established case of this lesion, compensated by hypertrophy of the right ventricle, serious obstruction to the outflow of blood from the pulmonary veins may go on for years, though the raised pressure in the pulmonary circulation may be evidenced by high accentuation of the pulmonary second sound during life, and by tortuous dilatation of the pulmonary capillaries seen after death. Nor can it be supposed that it is merely the rapidity of the onward flow maintained by the hypertrophied right ventricle that prevents the oedema appearing; for even when the right ventricle has failed and systemic dropsy set in, the latter may, as we have seen, persist weeks and months before any sign of pulmonary oedema appears. In view of this everyday experience it is difficult to understand what form of cardiac failure can take place in pneumonia which is to produce instantaneous and abundant oedema in the lungs without a trace of leakage from the systemic vessels.

Oedema of cardiac origin, further, as we see it in the later stages of valve disease of the heart, begins, as might be expected, in the most dependent parts, and thence extends upwards; but this is by no means always the case in pneumonic oedema, the signs of which may sometimes be detected in the scapular regions, or even in front, as early as at the base.

And, finally, I have satisfied myself in more than one actual case of severe pneumonia that signs of oedema have begun to appear before any appearance whatever of failure of the heart was observable.

In truth, I have long since found reason to believe that the theory of death from cardiac failure, which bulks so largely in clinical descriptions of pneumonia, is, to a considerable extent at least, founded on a misreading of clinical facts. Whenever I have myself had the opportunity of witnessing the terminal stage of a pneumonia it has been a respiratory and not a cardiac death that I have seen. Whenever the fatal stage has been sufficiently prolonged to allow of adequate physical examination, signs of oedema have preceded instead of following anything that could be called paralysis of the heart. What giving way of the heart or dilatation of its cavities takes place in pneumonia is, in my belief, more often a consequence than a cause of the lesions of the respiratory apparatus, the consequence, it may be, of advancing consolidation, it may be of bronchitis associated with consolidation, but in most cases of oedema attacking those portions of the lungs which have escaped inflammatory consolidation.

It is true that in a severe case the end sometimes comes on with startling rapidity, but we know how rapidly in many other instances oedematous fluid may increase once transudation has begun. In pneumonia we have less need to marvel at the rapidity of the oedema if, as seems reasonable to suppose, it is not of mechanical origin at all, but a direct effect of the pneumonic poison on the walls of the pulmonary capillaries. It is true also that in the most rapid deaths, little or no indication of respiratory distress is seen; but this by no means excludes death from asphyxia. Under No. 5,444, in our museum, you will find a lung taken from a recent case of mine in which death occurred rather suddenly, during a

moderate attack of haemoptysis, six or eight ounces only of blood being coughed up. At the necropsy the cause of death was plain; most of the secondary bronchi were plugged with fresh blood clot, and the lungs were in a state of forced distension behind them such as occurs during an attack of asthma. That death occurred from pure asphyxia was obvious, but I was assured in this case that the patient sank quite quietly, without any choking or appearance of suffocation. It is cases similarly rapid in their fatality that are especially attributed to cardiac failure, but their phenomena during life are equally compatible with the hypothesis of a death from the rapid occurrence of the oedema which is almost invariably found in the lungs after death.

I am labouring this point not merely as an interesting item of pathology, but as a matter of grave practical importance. The fear, exaggerated as I think it, of cardiac failure in pneumonia deters many medical men from that systematic examination of the back of the chest which, from my point of view, is necessary to descry the approach of the most serious danger to life. In dealing with a severe case of pneumonia the duty of repeated thorough examination of the chest, not merely for the purpose of defining the limits of consolidation but with the equally important end of knowing the state of the unconsolidated portions, cannot, I think, be too strongly insisted upon.

These are but two out of the many conditions of fatal disease in which oedema of the lungs plays a part in the closing scene. The frequency with which this condition is met with in necropsies has long been noted, though the tendency has been to overlook or to ignore its clinical importance and treat it as an inevitable and irremediable terminal phenomenon. Our careful pathologist, Dr. Salusbury Trevor, at my request recently ran through the records of 228 consecutive necropsies from January 1st to November 26th of the present year. He found that in no less than 124 oedema of both lungs, and, in 8 more, of one or other lung, had been discovered in such amount as to preclude the possibility of its being due to *post-mortem* oozing.

I cannot in the limited space of a single lecture discuss all the classes of disease in which this appearance was found, or attempt to determine in each the part which it may have played in the mechanism of death. One class of case only I will ask your attention to to-day in addition to those we have already considered; it is a class of case commoner even than heart disease or pneumonia. Simple as it may seem to assume that in a fatal case of bronchitis death has ensued owing to asphyxia from obstruction of the bronchial tubes, it is doubtful whether, except in very young children, death ever occurs precisely in that way. In more advanced life the reserve power of the respiratory system is very great, and bronchitis, perhaps, seldom occludes the entire system of tubes in equal degree. In children, catarrhal occlusion of the small tubes tends to induce collapse of the air sacs from the impediment it offers to inspiration; in adults inspiration seems rarely to be prevented to the same extreme degree, and the result on the air sacs is more often to produce distension of them owing to impeded expiration. But vesicles distended with air alone are not often seen in the *post-mortem* examination of a fatal case; over the greater part of the affected lungs the pulmonary tissue will be found to contain very little air, and a great deal of thin clear fluid. The lung pits on pressure; and the condition, in short, is again seen to be one of oedematous effusion. Now, in bronchitis there can be no practical question whether the oedema is an *ante-mortem* or *post-mortem* phenomenon. Its onset is gradual, and the signs of its progress are plainly discernible, not merely for hours, but for days and even weeks before a fatal termination, and for days and weeks also in many cases that do not terminate fatally at all. The course of a severe bronchitis in an elderly person (it is mainly at the extremes of life that the fatality of bronchitis is seen) often resolves itself into a combat between the physician and the oedema. I have many times warned you against the habit so prevalent in hospitals of passing over cases of bronchitis as "ordinary" or "uninteresting." The treatment of bronchitis forms a large proportion of general medical practice; no two cases of bronchitis are exactly alike, and every case invites individual study. The degree of emphysema present, the amount of available expansion, the presence of signs of bronchial dilatation, the probability of fibrous thickenings or of pleural adhesions, the condition of the right heart, the size of the tubes affected, the degree, if any, of bronchial spasm, all are questions very pertinent to the prognosis and treatment: but the most important point of all is to be quick to detect

the presence, among rhonchi and *râles* ascribable to bronchial secretion, of those sharper, clearer broken sounds that, as in our previous illustrations, indicate the onset and progress of oedema. The detection of them in bronchitis is more difficult, perhaps, than in any other form of disease, as *râles* of another kind so constantly accompany them; but with adequate practice it is easy to avoid error in most cases, particularly if the direct method of auscultation, without the intervention of a stethoscope, is habitually employed for the bases. In the absence of oedema and with evidence of sound heart tissue, the prospects of recovery in bronchitis are always good; the advent of signs of oedema indicates a further stage of graver import in the case.

I have thus restricted my illustrations almost entirely to the single subject of oedema. I have not even attempted to enlarge on the passive pleural effusions, passive pneumonias, and insidious broncho-pneumonias that play also a large part in the pathology of death, and that the careful examination I have urged of the pulmonary bases may also bring to light. The chief lesson I have sought to impress on you is the necessity of careful and repeated examination of these regions in cases of serious disease. Clinical mistakes, said Sir William Jenner, are more often due to neglect of examination than to lack of knowledge. When, therefore, your patient's countenance changes, when his complexion grows pale and dusky, when he becomes drowsy and apathetic, and his pulse begins to flag, do not content yourself with folding your hands and saying "toxaemia," or "collapse," or "asthenia," or "cardiac failure," but examine with care, and ascertain whether there may not be some condition involved that admits of treatment. The first three of the imposing terms I have cited are mere cloaks for ignorance, the last is more likely to be wrong than right. Unless its walls are the seat of fatty degeneration, the heart is a much more resistant organ than it is the fashion to admit, and has very little tendency to stop unless there is some strong hindrance to its continuing its work. Weakness of the pulse is not necessarily an indication that the heart is failing; it may equally be the result of deficient supply of blood to the cavities of the left side. A fatal end directly due to the heart is the exception rather than the rule in disease; in the majority of cases the issue of life and death is determined in the bases of the lungs.

The administration of "expectorants" still forms the staple of the current treatment of bronchial catarrh. Drugs that augment the fluidity of bronchial secretion or increase the activity of the expulsive cough are rationally indicated in dealing with such an affection. For ridding the lungs of oedematous fluid "expectorant" action is no less obviously useless; oedematous effusion is nearly always a thin fluid, and, as I have pointed out, it is in a situation from which it cannot be expelled by coughing. The clearing of the air sacs is chiefly the work of the absorbent vessels. How much of the task of absorption may fall on the veins I will not attempt to define; it seems at least reasonable to assume that the bulk of it falls on the pulmonary lymphatics, which deliver into the thoracic and right lymphatic ducts. These ducts discharge, I may remind you, into the great veins at the root of the neck, and the efficiency of their discharge is therefore affected by the degree of blood pressure in these veins, which in its turn depends on the efficiency of the right side of the heart. A careful investigation of the right side of the heart is, therefore, an important preliminary to the treatment of oedema of the lung, particularly as the right ventricle, even if previously healthy, is liable to give way before the strain of any sudden and severe obstruction to the pulmonary circulation. The signs of dilatation of this ventricle, undue extension of dullness to the right of the sternum, displacement of the apex beat to the left, the presence of a heaving impulse over the middle of the heart, coupled with blunting of the apical beat, or the transference of the most marked impulse to the epigastrium, must be looked for. Of even greater importance is it to note the presence of any direct evidence of obstructed venous circulation, such as may be afforded by distension of the jugular veins.

If these signs are present and the conditions are at all urgent, venesection should be without hesitation resorted to. The letting of blood may be practised without fear; but I am not convinced that the large amounts sometimes recommended are really necessary. The removal of 10 or even 6 oz. of blood has in some cases seemed to me to do all that is necessary. In very urgent cases, however, it is safe to go to 15 or 20 oz. at once.

The relief afforded by venesection is often very marked; not always as speedy as would be the case were the relief of the right ventricle the sole question at issue, but as speedy as can be expected on the assumption that absorption of some portion of the oedematous fluid is the actual means of relief.

Failing venesection, other less efficacious methods of reducing the venous pressure may be resorted to. The application of six or eight leeches over the front of the chest is in some cases attended with good results, though less marked than those furnished by venesection. We may, further, use with effect free watery purgation, by means of elaterin, jalap, or gamboge, or the synthetic purgative compound that has been recently introduced into medicine. Diuretics are practically useless; a hot-air bath is not always applicable, and the only other really efficient sudorific—pilocarpin—is contraindicated by the probable state of the heart. I will mention, also, dry cupping, as it is recommended by some authors, though my own experience of it is insufficient for me to give a definite opinion of its merits.

When marked dilatation of the right ventricle has existed, some damage to the left is nearly certain to have followed. On account of both ventricles bleeding should be followed up by the administration of digitalis, digitalin, strophanthus, convallaria, or squill, to aid in restoring the tone of the stretched cardiac wall. None of these drugs are likely to be of avail unless any serious engorgement of the right side that may exist has previously been relieved by more direct means; but in lesser degrees of dilatation, unaccompanied as yet by urgent symptoms, one or other of them may suffice to overcome the cardiac difficulty without resort to the removal of blood.

Though isolated doses of alcoholic liquors, particularly brandy in hot water, are often of service as temporary stimulants, particularly in the early hours of the morning, when the forces of the heart and of the respiration are at their minimum, the continuous administration of alcohol when oedema is present in the lungs has always seemed to me useless, if not actually injurious. Oxygen will, of course, find a place in the plan of treatment. The reasons for employing it are so obvious that I need not enlarge on them. The effects, it is true, are only palliative; but then a mere postponement of complete asphyxia may be of cardinal importance in the case. The use of atropine and ammonia as stimulants to the respiratory centre is at least indicated by theory if not fully confirmed by practice, and may be employed without risk. I was in the habit of administering potassium iodide in these cases at one time, on account of its alleged "absorbent" properties, but have seen no reason to think that it has any good effect.

One drug I have yet to mention, and that, I believe, the most important of all. I have so often seen marked and speedy improvement, accompanied by a corresponding clearing up of the characteristic signs, follow the administration of strychnine by hypodermic injection in cases of pulmonary oedema that I can scarcely suppose the connexion to be accidental. How the strychnine acts I will not pretend to say; its effects are equally observable in slight cases and in severe ones, in cases apparently independent of cardiac dilatation, and in cases accompanying it. I have for some years past regularly resorted to this mode of treatment almost as a matter of routine as soon as any signs of serious oedema of the lungs appear, to whatever cause they may be due.

THE COCAINE HABIT IN WASHINGTON.—The superintendent of the Washington police force has recently reported that the cocaine habit is greatly on the increase among the lower classes of that city, and urges that vigorous measures should be taken at once to check the evil. He says: "It (cocaine) is bound to become a destructive agency unless something is done to prevent its sale by druggists. Two years ago the drug was hardly known, but the habit has grown, and the wreck of lives which accompanies the use of this deadly drug and its sister drugs, morphine, chloral, and other pharmaceutical preparations, and even patent medicines, is becoming apparent." At present the police are almost powerless in the matter. Mr. J. M. Robinson, of Indiana, a member of the House of Representatives, intends to introduce a Bill for the regulation of the sale of poisons in the district of Columbia. The Bill has the approval of the Medico-Legal Society of Columbia, and the measure is in harmony with recommendations made by the coroner, Dr. J. Ramsay Nevitt, in his annual report to the Commissioners of the District of Columbia.