This is partly the result of the prone position of the patient in bed and of weakness of expectorating power, and partly owing to the suction power of the middle portions of the lung being stronger than that of the apex. Hence the secretion from an apex cavity, instead of being discharged through the wind-pipe and mouth, descends into an adjoining bronchus, reaches the terminal bronchioles and alveoli, and sets up fresh tuberculous disease in the neighbourhood. A cuboidal mass of tubercle is formed as the result of the infection, which again breaks down and is expectorated, and thus another cavity is formed. These secondary cavities follow the lines of certain bronchi, principally where the suction power on coughing is greatest, and the principal secondary cavities are formed in the dorso-axillary region, the mammary regions, and at the base of the lung, and are remarkable for being devoid of lining membrane. The dorso-axillary region is the commonest spot for a secondary cavity to commence in and should be carefully examined in chronic cases, but a large number of secondary cavities are also detected in the mammary region. The basic cavities are not so common. Secondary cavities are unfavourable to a good prognosis, as they seldom contract.

AMPHORIC CAVITIES.

In some patients tuberculization is followed by so extensive an amount of excavation that the presence of air in the cavity gives rise to the phenomena of amphoric breathing and metallic tinkle, which latter is the echo of drops of fluid falling into the cavity from its walls and bronchi.

These cavities are diagnosed from pneumothorax by the absence of the fluid in the pleura and its characteristic signs, and by the different position of the auscultation sounds, though there are cases of localized pneumothorax where the diagnosis is very difficult and often impossible.

CONTRACTING CAVITIES.

The phenomena these give rise to may be traced step by step by physical signs. Cavities contract sometimes rapidly, and I have known considerable sized cavities contract in three months, but as a rule the process is a slow and gradual one, depending on the amount of fibrosis in the lung and the reacting power of the patient against the ravages of the tubercle bacillus. The chief signs of a contracting cavity are:

1. Flattening of the chest walls overlying the cavity, and often contraction of the chest circumference of the side.
2. Displacement of neighbouring organs.

When the contracting cavity is on the left side, the left lung is retracted towards its root, and the right lung becomes hypertrophied, and is drawn across the median line of the sternum, and may be traced through percussion by a line of resonance reaching half-way across the clavicle. The heart is raised, its apex being tilted towards the axilla, and sometimes it describes an arc of a considerable sized circle, of which the centre is the fixed portion of the heart and arteries. The heart's impulse may be felt first in the fourth space to the left of the nipple, and even traced into the third space, always tilted towards the axilla. The stomach is drawn up, and the note is to be distinguished quite high up in the thorax.

When the cavity is in the right lung, the heart is generally drawn to the right and under the sternum, the liver is raised, and the left lung is drawn across the median line, and reaches half-way across the clavicle, or even further. The lung on either side contracts towards a fixed point, sometimes towards a spot where the pleura is adherent, but more commonly towards the root of the lung; and the cavernous sounds, at first becoming less distinct in the first and second interspaces, become more so in the suprascapular and interscapular regions, where they linger some time, and gradually disappear. Cough and expectoration may cease, or the latter sometimes becomes fetid from retention through narrowing of the bronchus. Occasionally the contraction of cavities causes even greater changes, and the shoulder of that side is depressed, the spine being curved towards the contracting side. The worst of it is that contraction of the cavity in one lung does not prevent the formation of fresh tubercle in the opposite lung.

DIAGNOSIS OF CAVITIES.

The diagnosis of tuberculous cavities from bronchiectasis is based on several points of distinction.

First, on the age of the patient and on his different appearance in the two diseases. Children and middle-aged people are liable to bronchiectasis, but as a rule not young adults, which is the class most frequently attached by tuberculosis.

Patients with bronchiectasis, except when suffering from septicaemia, show no sign of wasting or anaemia, but generally have a healthy exterior and are commonly gaining flesh.

Secondly, on the appearance and examination of the sputum, which in the case of bronchiectasis is frothy and mixed with pus and more or less offensive, and, though containing numerous septic organisms, is free from the tubercle bacillus.

Thirdly, on the character of the cavernous sounds and their locality. The majority of bronchiectatic cavities are basic, whereas tuberculous cavities are generally at the apex. Bronchiectatic cavities may, however, be detected in the first and second interspaces, where they are always nearer the median line than tuberculous cavities, and though they give rise to noisy, jarring, cavernous sounds, they are never audible over such extensive surfaces of the thorax as in tuberculous cavities. The percussion sound is not as a rule dull, but resonant from the accompanying emphysema.

The Harveian Lectures

ON

THE TREATMENT OF SOME ACUTE VISCERAL INFLAMMATIONS.

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LECTURE I.

ACUTE RHEUMATIC CARDITIS AND PERICARDITIS.

The treatment of acute cardiac inflammation is, in the great majority of cases, the treatment of an infection with the organism of acute rheumatism, and we have to consider what therapeutic agents, general and local, are available to assist us.

First and foremost, we must determine the value of sodium salicylate as an internal remedy in acute rheumatism. It is believed by some that the value of salicylate in the treatment of rheumatism is limited to the reduction of temperature, the relief of pain, and the diminution of arthritis. But a very short experience in the wards of a hospital for children suffices to show that this view is quite inadequate. For in the rheumatism of childhood arthritis is often absent altogether, and pain may be slight, while cardiac disease is severe. Yet the good effects of salicylate in children are quite as well marked as in adults. The temperature falls, the general condition improves, torpidity gives place to a bright expression, and the cardiac state is ameliorated, the area of dullness tending to diminish, and the strength of the ventricular contraction to increase.

Observation of the effects of salicylate in the treatment of acute rheumatism both in children and in adults has led me to the conviction that it is as truly antirheumatic as quinine is antimalarial or mercury antisymphilitic. But it must, of course, be given in adequate doses and its use must not be too soon relinquished. For an adult 20 gr. of sodium salicylate with 40 gr. of sodium bicarbonate should be given every two hours during the day and every four hours during the night. For a child of 6 to 10 years old 10 gr. of salicylate and 20 gr. of bicarbonate at the same intervals; this amounts to a daily dose of 100 gr. of salicylate. After a day or two 15 gr. of the salicylate with 30 gr. of bicarbonate may be given, and if necessary this may be increased to 20 and 40 gr. respectively, the total daily amount of salicylate being 150 or 200 gr. Children require proportionally larger doses than adults, for in them the rheumatic infection is much more intense. It is rare for an adult to die from his first rheumatic attack, but this disaster is by no means uncommon in childhood. Fortunately children usually bear salicylate well; in them it seldom causes much vomiting, often none at all, and they hardly ever complain of the deafness, tinnitus, and headache which are troublesome in adults, nor do they often manifest the mental symptoms and the tendency to delirium which are sometimes caused by the drug in later life.

There is, however, one effect occasionally produced by salicylate which demands special mention, for it is a sym-

ptom of danger, and its occurrence should lead to an immediate discontinuance of the drug. It is a marked deepening of the inspirations (apart from pericarditis), and resembles the "air hunger" of diabetic coma. In diabetes an incipient air hunger may sometimes be arrested by the administration of large doses of alkalies, and in the only fatal case of salicylic air hunger which has come under my own observation the salicylate had been taken without any added alkali. It seems probable that the symptom is due to an action on the respiratory centre caused by an excess of acid. An addition of sodium bicarbonate in quantity equal to twice that of the salicylate in each dose seems to be usually sufficient to prevent the occurrence of this symptom.

If for any reason in child or adult it seems necessary to discontinue the salicylate, it should only be suspended for a few hours; then it should be again given in a smaller amount, and the dose gradually increased. It is almost always possible in this way to accustom the patient to the drug, and to get him to tolerate much larger doses than at first produced disturbance.

CASE I.—A woman, aged 25, suffering from mitral stenosis, with high temperature and fresh endocarditis so severe as to suggest a diagnosis of malignant endocarditis, vomited and became delirious on 20-gr. doses of salicylate. By omitting the medicine for a few hours, and then giving smaller doses at first, she tolerated the remedy well, and it was soon possible to increase the amount as far as 30 gr. every two hours. The threatening signs and symptoms disappeared, and when she was discharged only a tranquil stenosis remained.

[In severe cases large doses may be necessary, as much as 30 gr. every two hours for an adult, occasionally 25 or even 30 gr. for a child. It is very desirable to continue the use of the drug for a considerable time after the subsidence of the rheumatic symptoms. Too early discontinuance of the remedy, or a too great reduction of the dose, is often in adults, and still more frequently in children, quickly followed by a relapse. Too great timidity in the employment of salicylate may cause an adult patient additional pain which might have been avoided; in a child it may be the cause of a cardiac disaster.]

CASE II.—A boy, aged 5, with a greatly dilated rheumatic heart, was treated with 8 gr. of sodium salicylate and 16 gr. of bicarbonate every two hours for sixteen days, and manifested decided improvement. The frequency of the dose was then reduced to every four hours, that is, the daily dose of salicylate was reduced from 80 gr. to 48 gr. Five days later, while the smaller dose was still being taken, a relapse occurred, with increased cardiac dilatation, and this led to his death three weeks later.

CASE III.—A woman, aged 52, suffering from rheumatic arthritis in many joints, and with loud pericardial friction, was treated on July 10th, 1903, with 200 gr. of sodium salicylate daily, with 400 gr. of sodium bicarbonate, and an icebag was applied over the heart. Four days later the arthritis had disappeared, and the rub was much less loud, though the temperature had fallen only from 103.5° to 102°. But she complained so much of headache, deafness, and tinnitus that the medicine was discontinued altogether for two days. On July 16th it was again given, but in smaller amount—120 gr. of the salicylate and 240 gr. of bicarbonate. The temperature continued to fall for two days longer, and on the 18th was only 99°; but on the 19th, five days after the temporary suspension of the remedy, it rose again to 100.2°, and continued at this height on the 20th, with a further rise on the 21st to 101.8°. It seemed evident that a relapse of rheumatism was coming on, which the amount of salicylate and bicarbonate then being taken was insufficient to prevent. With a view of testing the question whether it was the salicylate that was really necessary, or whether an increased dose of the alkali would suffice, the daily dose of this was raised to 360 gr., the amount of salicylate being still only 120. The temperature rose on that day to 103.4°, and the next day (23rd) to 103.9°, and fresh rheumatic pain was then felt in the right hand. On the 24th the metacarpophalangeal joints of this hand were red and swollen. It was clear that the increase in the amount of alkali had failed to meet the need. The dose of salicylate at first given was therefore resumed, 200 gr. daily with 400 of bicarbonate. Next day (25th) the redness and swelling of the fingers had subsided, but on the 26th a fresh pericardial rub was heard, which was louder and harsher on the 27th. On the 28th it was very much less, and the temperature had fallen to the normal. From July 26th to August 8th she took 160 gr. of salicylate and 320 of bicarbonate; but as the temperature again rose to 99.5° on the 8th the doses were again increased to 200 gr. and 400 gr. There was no further recurrence of rheumatism, and she now took without difficulty the same doses as had at first caused disturbance. The temporary omission of the medicine and the smaller subsequent dose were clearly responsible for this fresh rheumatic outburst in joints and pericardium.

The employment of salicylate in sufficient doses for a sufficiently long period is much hindered by a tradition current amongst us that this drug is a "cardiac depressant." This doctrine has been maintained by some physicians of great authority as clinical observers, and especially they have taught that when pericarditis occurs in a patient suffering from rheumatic fever the salicylate ought to be stopped lest it should depress the heart still more. Now it is clear that a belief of this kind must have been founded on experience of a tendency to failure of the pulse and weakness

of the first sound of the heart observed in rheumatic patients while taking salicylate. But one may fairly ask whether it is quite certain that such occurrences are really the effect of the drug, whether they may not be due to some other cause? In the first place they may conceivably be due to impurities of the drug as administered. Especially in the early years of its use, when a sudden demand arose for large quantities of the new remedy, it is quite likely that adulterations due to imperfect methods of preparation may have caused toxic symptoms, the memory of which remained in the minds of those who witnessed them and gave to the drug an evil reputation which has clung to it ever since. The sodium salicylate now obtainable is carefully prepared and purified and is free from impurities of cresols, etc. That obtained from oil of wintergreen is more than ten times as costly, so that its employment in large doses is impracticable.

But there is another and a more important fact which has been the main cause of the belief in the depressing effect of salicylate. It is unquestionable that rapid extension of the dullness of the left ventricle with enfeeblement of the first sound and of the pulse may occur in rheumatic patients who are taking salicylate. But is this due to the drug or in spite of it? Has it caused these symptoms or merely failed to prevent them? Can they be due to the disease itself?

Acute rheumatism produces, apparently in all cases of the disease, more or less dilatation of the left ventricle, with a tendency to diffusion and weakening of the impulse and enfeeblement of the first sound. I drew attention to this fact in a paper on acute dilatation of the heart in rheumatic fever, followed by a joint paper with Dr. Poynton on the same phenomenon as observed in the rheumatism and chorea of childhood, both published in the *Transactions* of the Royal Medical and Chirurgical Society for 1898. These observations have not, so far as I am aware, been controverted, and they have been confirmed by subsequent experience.

In the mildest case of subacute rheumatism the dullness of the left ventricle hardly ever fails to reach the nipple line; usually it extends a fingerbreadth beyond it. It may attain to two fingerbreadths to the left of the nipple line, without any murmur, and it may return to the normal as the attack subsides. This dilatation is due, no doubt, to the toxic action on the cardiac muscle of the toxin produced by the rheumatic organisms, frequently also to the actual presence in the heart wall of the organisms themselves. We can better appreciate the probable effect of the toxin by remembering Dr. Gaskell's experiments recorded in the third volume of the *Journal of Physiology*. Dr. Gaskell found that a dilute solution of lactic acid caused relaxation and "extreme dilatation" of the frog's ventricle, and finally diastolic standstill; it caused relaxation also of the vessels. On the other hand, a dilute solution of sodium hydrate caused gradual progressive contraction of the ventricle until it remained persistently fully contracted, and a similar contraction of the arterioles. He investigated also the action of various drugs, and found that some of them acted upon the ventricle like soda, others like lactic acid.

It seems almost certain that the toxin produced by the rheumatic cocci acts upon the ventricle in a manner similar to that of lactic acid; and it is of interest to remember that cultures of these organisms, even in an alkaline medium, always become acid; also to remember the belief of many former clinical observers that the poison of acute rheumatism is lactic acid itself. Important evidence on this question has recently been brought forward by Dr. Ainley Walker and Mr. Ryffel in a report to the Scientific Grants Committee of the British Medical Association.¹ They state that the "micrococcus rheumaticus" produces formic acid in very considerable quantity, and that this acid is not only present in the filtered cultures of the organism, but it can also be extracted from the bodies of the micro-organisms themselves. From these, after repeated washings to remove adherent traces of the culture fluids, they have obtained appreciable amounts of formic acid. It follows that the processes concerned in its production proceed within the body of the micro-organisms themselves. From a litre of culture fluid they were able to obtain about half a gram of formic acid. In addition to formic acid, the cultures and also the washed micrococci contain at least one other acid of the fatty acid series, probably acetic.

They also succeeded in obtaining formic acid from the tissues of a rabbit suffering from acute arthritis due to the inoculation of this micro-organism.

They state, also, that while from normal urine formic acid is absent or occurs in traces only, formic and probably another fatty acid are present in appreciable amounts in the urine

during the course of acute rheumatism, and they have some evidence that under the salicylic treatment of rheumatism the amount of formic acid in the urine is reduced.

With regard to the origin of the formic acid, they believe it to be formed by the micro-organism by the oxidation of sarcolactic acid into acetic and formic acids according to the equation:

They add two facts of much importance. They have observed that the micrococcus rheumaticus has a haemolytic action upon red blood corpuscles greater and more rapid than that of any other streptococcus which they have yet examined, which explains the rapid and considerable anaemia of acute rheumatism. They have also succeeded in isolating an albumose from cultures of the organism grown in albuminous fluids free from albumoses, which on injection into animals (guinea-pigs and rabbits) rapidly produces marked pyrexia, and leads to an increase of temperature of three or more degrees Fahrenheit, reaching on one occasion 105° .

These observations of Dr. Ainley Walker and Mr. Ryffel are a justification of the ancient practice of treating acute rheumatism with alkalies. Before the specific action of the salicylate was discovered the alkaline treatment of rheumatism was the most successful, though at that time potassium salts were chiefly used and in comparatively small quantities. But sodium salts can be given much more freely, and Dr. Gaskell's observations on the frog's heart seem to indicate that they also tend to cause contraction of the ventricle. I think that there is some clinical evidence that this is really the case when they are freely employed in rheumatism.

If, then, the rheumatic toxin always tends, more or less, to dilate and enfeeble the left ventricle, it is not surprising to find that in some cases a "cardiac depression" may manifest itself somewhat suddenly, after the disease has lasted for some days, or even a week or two. If the patient is taking salicylate when this occurs, the drug is almost certain to receive the discredit of symptoms really due to the disease itself. But strong evidence as to their actual cause can be adduced from analogy, for a similar acute dilatation and enfeeblement, but of a much more dangerous kind, is met with in some other diseases, notably influenza and diphtheria.

I pointed out, in an address on Acute Dilatation of the Heart in Diphtheria, Influenza, and Rheumatism, delivered before the Manchester Medical Society in 1900, and published in the BRITISH MEDICAL JOURNAL for January 5th, 1901, that in all these diseases an acute cardiac dilatation is not uncommon, and that it may cause fatal syncope in diphtheria and in influenza.

The symptoms before death are pallor, sometimes vomiting, and feeble (perhaps irregular) pulse. The physical signs are a rapid increase in the heart's dullness to the left, which may amount to an additional fingerbreadth in a few hours, diffusion and weakening of the impulse, with weakening of the first sound and of the pulse wave. After death extensive fatty degeneration of the cardiac muscle is found. The symptoms of an acute dilatation in rheumatism are less serious than those of a similar condition in diphtheria and in influenza, probably because there is less destruction of the cardiac muscular fibre. The acute dilatation of rheumatism rarely, if ever, produces a fatal syncope, but it may give rise to marked pallor and to obvious changes in the pulse. In diphtheria an acute dilatation may occur at any time during the six weeks following the onset of the disease. In rheumatism it may occur as late as two or three weeks after the onset, perhaps later.

CASE IV.—A woman, aged 29, was suffering from her first attack of rheumatism. It was of a subacute type, with but little arthritis; the cardiac dullness extended to the nipple line but not beyond it, and there was no murmur. She was treated with sodium salicylate. After she had taken this for a fortnight with benefit the house-physician one day observed that the pulse was unusually weak. He percussed the heart at once, and found that the dullness of the left ventricle now extended one fingerbreadth beyond the nipple line, and a short systolic murmur could be heard. I confirmed these facts on the following day. Here was a case in which it might have been suggested that the salicylate had caused cardiac failure. But instead of omitting the drug I ordered it to be given more frequently, and placed an icebag over the heart. Immediate improvement followed, and in a few days the cardiac dullness was again normal, and the faint murmur had disappeared.

In diphtheria it cannot be doubted that an acute cardiac dilatation is due to the disease itself, and not to any drug that has been administered. I am convinced that in rheumatism also it is the disease itself which is responsible for

cardiac failures, and that they are not due to the salicylate. Indeed, they are probably in many cases an indication that an insufficient amount of the drug has been given.

If more care were taken to determine the exact size of the left ventricle by careful light percussion at the commencement of treatment and daily afterwards, an acute dilatation would be at once detected. There is little difficulty in this determination if the fingers only and not any "pleximeter" are used for percussion, if the part of the finger percussed is the terminal phalanx, and the other phalanges are not allowed to rest on the thoracic wall, and if the percussion stroke is always light. It is a primary duty to determine this matter with care whenever a practitioner is called on to attend a case of diphtheria, of influenza, or of rheumatism. It is, I believe, the neglect of this which has caused salicylate to be considered a cardiac depressant, and thus has done much mischief to rheumatic patients, for it has deprived them of a necessary drug. If, then, salicylate is not a cardiac depressant, and if it is genuinely antirheumatic, it is surely specially necessary in order to counteract the most deleterious action of rheumatism—its tendency to cause cardiac inflammation. The worst type of rheumatic carditis is associated with pericarditis, and it is here that the drug is most urgently needed. To withhold salicylate from a rheumatic patient, child, or adult, unless some equally efficacious medicine can be substituted for it, is, in my judgement, to do the patient a grievous injury. In medicine, as in ordinary life, sins of omission are more lightly regarded than sins of commission, but they may be quite as disastrous.

In support of my plea for the employment of larger doses of salicylate in the treatment of rheumatic conditions I may mention that I have lately found great advantage in the administration of large doses of salicylate and bicarbonate in many cases of chorea. The small doses of these remedies usually given in this disease (50 to 60 gr. a day) are far too small. If 100 gr. of salicylate as daily dose are given at once, and the amount rapidly increased to 150 gr., and if necessary to 200, very remarkable improvement often occurs, as I showed at the discussion on chorea at the Swansea meeting of the British Medical Association. After this discussion, a fact of interest was mentioned to me by Dr. McVittie, of Dublin, and he kindly allows me to relate it now. Through the mistake of a chemist, 2-drachm doses of sodium salicylate were taken by a patient three times daily for a week, with much benefit, and without the occurrence of any unpleasant symptoms.

Encouraged by this observation, Dr. McVittie prescribed for another patient suffering from chronic rheumatism, powders containing 2-drachm doses of salicylate, to be taken three times a day. The chemist to whom the prescription was taken thought that a mistake had been made, and distributed two drachms of the salicylate, as a total amount, among the twelve powders. The patient took these powders and was no better. Then he had the prescription made up by another chemist, who put into the powders the full dose ordered. In a week the man was well. In two subsequent cases of chronic muscular rheumatism Dr. McVittie observed complete relief of all pain follow the use of these powders, and no unpleasant symptoms were produced.

My conclusion is that sodium salicylate has certainly a marked antirheumatic action, that a considerable daily dose of the drug is essential to success, that each dose should be accompanied by twice as large a dose of sodium bicarbonate, and that the joint action of the two drugs should be maintained for some time after the subsidence of the rheumatic symptoms.

We have, therefore, in the free administration of sodium salicylate and bicarbonate an adequate method of meeting the causal indication in the treatment of rheumatic inflammation of the heart and pericardium. It remains to inquire whether we have also any means of directly diminishing the inflammatory action by local applications. The use of blisters over the precordial region was at one time a favourite method of treatment, and it is at least conceivable that the counter irritation of the overlying skin may tend to diminish the fullness of the vessels in an inflamed pericardium. But it is not desirable if it can be avoided to add to the sufferings of a patient who is seriously ill, and the local effect of a blister greatly impedes the necessary examination of the heart. Leeches applied to the cardiac area have a distinctly beneficial effect. They probably have some direct influence in draining the pericardial vessels, and they certainly cause a diminution of pressure in the right auricle, which is often of the greatest service. But this latter

effect can be equally well obtained by applying them to any other part of the thoracic wall, thus lessening the supply of blood through the azygos vein to the right auricle, and the subsequent examination of the heart will then not be impeded. The most convenient place for their application is over the lower anterior part of the right chest below the nipple level; it is always possible to arrest the flow of blood by pressure against the firm resistance of the ribs. But if the leeches are applied over the soft parts at the epigastrium there may be very considerable difficulty in arresting the flow.

The persistent application of cold to the precordial region by means of an icebag is a powerful means of repressing cardiac inflammation. In rheumatic pericarditis its effect is unmistakable, and often very striking. I have employed it in nearly every case under my care during the last eleven years, and have found it extremely useful. At first I was very cautious in using it, only applying it for a short time, lest the cold should further depress a heart already hampered by the pericardial inflammation. What led me to employ it was the observation that, where an icebag had been applied for some hours to a pneumonic lung, it was usually found that the dullness at that spot was less intense, and that there was a better air entry, indicating that the vascular congestion of the lung beneath the icebag had been lessened. I found also that the symptoms and physical signs of subacute inflammation of the appendix vermiformis were rapidly relieved by an icebag, and that the same was true of sciatic neuritis of recent origin.

In all these three cases the direct local influence of the icebag in repressing subjacent visceral inflammation seemed undeniable, and it occurred to me that possibly it might have the same beneficial influence in pericarditis. I soon found that the inference was correct, and that there was no danger of collapse if two precautions were kept in mind.

One of these precautions is that the patient be made quite warm, if necessary by hot-water bottles, before the icebag is applied to the heart, and that the warmth be maintained during the whole period of its application. The second precaution is that the right auricle shall not be allowed to be overdistended. A few leeches, applied as already recommended, are sufficient to fulfil this condition, and it is generally advisable to apply them before the ice is used.

If these two conditions are secured, the icebag may be used without fear in a case, however severe, of rheumatic pericarditis. With these precautions the local use of ice, so far from depressing the heart, has actually a tonic action. The feeble and diffused impulse of the left ventricle becomes localized and stronger; this effect is often very noticeable after the icebag has been used continuously for some days. The friction sound soon becomes less harsh, and is heard over a smaller area of the precordium. This is not due to increased exudation, for the area of precordial dullness tends to diminish if at the same time large doses of sodium salicylate and bicarbonate are being administered, the improvement may be very rapid, and the diminution of the dullness remarkable. It must be remembered that in rheumatic pericarditis the effusion of fluid into the pericardial sac is only a minor factor in the increase of the dullness; this is mainly caused by dilatation of the heart.

It is necessary to insist on this fact, for it is not yet generally recognized. Rheumatic dilatation of the heart without any pericarditis may give rise to an outline of precordial dullness, which is practically a triangle with curved sides, and with its apex upwards. When pericarditis is present also, a moderate amount of pericardial effusion tends to accumulate behind the dilated heart in the recumbent position, and does not much influence the shape of the dullness. The greatest effect of a moderate pericardial effusion on the dullness is produced in the second left intercostal space, in which a definite lateral increase of the dullness suggests strongly the existence of fluid in the pericardial sac. A large pericardial effusion will no doubt extend the cardiac dullness further in all directions, but it remains true that in rheumatic pericarditis the chief factor in the enlargement of the dullness is dilatation of the heart.

The outline of the precordial dullness on the right side is mainly indicative of the state of distension of the right auricle. This statement also needs to be specially insisted on, both on account of its value as a suggestion for treatment and because it is as yet by no means universally acknowledged that the condition of the right auricle can be determined by careful percussion, its enlargement in disease of the lungs or left heart watched and recorded, and its diminu-

tion after leeches or venesection easily appreciated. In the normal heart the dullness of the right auricle always extends about one fingerbreadth (rather less in a child) to the right of the sternum in the fourth intercostal space. This can easily be verified by any one who avoids all artificial pleximeters and percusses with a light hammerstroke, with the flexed terminal phalanx of a finger of his right hand, on the terminal phalanx of a finger of his left hand, firmly applied, taking special care to keep the other phalanges of the percussed finger and the whole of the rest of the left hand away from contact with the thoracic wall. Unless this precaution be adopted, pulmonary or gastric resonance is brought out at the same time as the truly cardiac and confuses the result. Lightness of percussion stroke also assists in obtaining the true cardiac note, for though the right auricle is overlapped by lung, the auricle with its contained blood gives a dull note which the thin overlying lung does not alter much, and at the border of the auricle there is a definite alteration in the note which careful percussion easily elicits.

In the normal heart it is usually difficult to detect any dullness in the third right space, but it is always definite and considerable in the fourth. In the fifth space the partial liver dullness complicates the result. It is of the greatest practical importance that the extent of the dullness in the fourth right space should be carefully determined in every examination of the heart, and no such examination is anything but fallacious unless this point has been carefully investigated.

In chronic disease of the lungs or left heart, as in emphysema, and in mitral stenosis, the right auricle is always dilated, and its dullness may extend to two fingerbreadths in the fourth right space: it is then easily detected in the third space also, and may there amount to a half or one fingerbreadth. In extreme cases it may reach even three fingerbreadths in the fourth space, one and a-half in the third and half a fingerbreadth in the second.

In acute pulmonary disease, as in pneumonia, and in severe bronchitis, an enlargement of the right auricle may be detected, often on the third day of the illness, sometimes even on the second. Acute rheumatic carditis affects at first and chiefly the ventricular muscle, and causes distinct enlargement of the cardiac dullness to the left, but in a first attack it is difficult to detect any affection of the auricle. But where pericarditis is present there may be marked increase of the auricular dullness. And this observation is of great importance from the point of view of treatment. For whenever the right auricle is acutely distended there is hurried breathing. This is usually the explanation of the dyspnoea which is acknowledged to be one of the symptoms of pericarditis; and this dyspnoea is a sign that the right heart is overstrained. In pericarditis, then, if there are rapid and deep inspirations and the dullness of the right auricle in the fourth space is found to amount to two fingerbreadths, the need for some removal of blood is evident. Sometimes leeches applied over the lower ribs on the right side will suffice, but in a severe attack in a robust subject venesection may be highly advisable. I have seen it give very great relief. The same is true in the acute distension of the right auricle in pneumonia, in acute exacerbations of chronic bronchitis, and often in the later stages of mitral stenosis. Dyspnoea and the physical evidence of dilated right auricle are the indications for bleeding, which in these circumstances is a remedy of priceless value, capable of giving immediate relief impossible by any other means.

The first step, then, in the local treatment of rheumatic pericarditis is the accurate determination of the amount of dullness in the fourth right interspace, and the relief of a distended right auricle by leeches, or in an adult by a venesection removing from 4 to 8 oz. of blood. It may be well to follow this by cardiac stimulation by means of a hypodermic injection of strychnine. Two minims of the official solution may be used for an adult, $\frac{1}{2}$ or 1 minim for a child. The injection may be repeated after three hours. Meanwhile, at least two hot-water bottles should be placed in the bed so as to make the patient's lower limbs thoroughly warm. When this has been accomplished (and not before) an icebag large enough to cover the whole precordial region and about half filled with small lumps and fragments of ice should be gently laid over the heart as the patient lies on his back. If there is much tenderness the icebag may be at first suspended so as only just to touch the skin, but the anodyne effect of the cold will soon allow of its more thorough application. The icebag will not by its weight increase the difficulty of breathing if the right auricle has been first relieved.

The patient soon recognizes the beneficial influence of the ice. Even young children, after the first few minutes, become easily accustomed to the cold, and will not consent to its removal. When it has been removed for an interval they will sometimes ask that it may be reapplied. A boy of 7 under my care at St. Mary's refused to allow his icebag to be taken away. To the inquiry, "Why not?" he answered, "Because I like it." To the further inquiry, "Why do you like it?" he replied, emphatically, "Because I do."

The chief difficulty about the application of the icebag is to secure it from shifting its position when the patient turns in bed. This may be accomplished by passing the screw top of the icebag through a hole in a binder which is loosely passed round the chest and secured by safety pins. Sometimes it may be desirable to prevent its slipping downwards by securing it by a light bandage from behind the patient's neck. An excellent method used at the Hospital for Sick Children is by a vest with armholes, and a third hole over the cardiac region, made of domett, and fastened round the neck with a tape, and below the icebag by a safety pin.

The outline of the precordial dullness should be marked on the skin in blue for the guidance of the nurse. The icebag will require to be refilled about every hour and a-half, the hot-water bottles every three hours.

It is well to have a second icebag in use, so that it can be filled and applied as soon as the first is removed. In filling the icebag, be careful to press out the air as much as possible before screwing on the top. The hot-water bottles should not all be removed from the bed at the same time, unless others are at once substituted. The patient's temperature should be taken every two hours, also his pulse-rate and respiration-rate, and the result recorded on a chart. The nurse must see that the icebag does not leak, that its top is firmly screwed down to the "washer," and that it is surrounded by cotton wool or a soft towel to absorb the moisture from the air which tends to condense on its surface. But nothing should intervene between the icebag and the patient's skin.

When the nursing can be thoroughly trusted, as in a hospital, the icebag may often, with great benefit to the patient, be applied continuously for many days, provided that the condition of the right auricle be carefully watched, and the lower limbs be kept thoroughly warm. Occasionally it may be wise to remove it for a few hours during the night, especially between midnight and six o'clock in the morning. If the nurse has had little experience of children, or is not accustomed to the use of the icebag as here recommended, it will be safer to apply it only during the daytime. If no skilled nursing is available, the treatment may still be used during the daytime if the mother is intelligent and careful, and the practitioner explains to her the use of the clinical thermometer, and tells her precisely what to do. If the practitioner is in doubt whether the icebag ought to be continued, let him order its removal for one hour, and thereafter its application for two hours, and so on.

It is sometimes possible to continue the application of ice to the precordial region even though the temperature have fallen below the normal. A child under my care at St. Mary's some years ago, whose pericarditis had improved under this treatment, was found one evening to have signs of pneumonia at the base of one lung. Twice before I had met with this in young children suffering from pericarditis and treated with ice; in each case when the pneumonia appeared I removed the icebag from the heart, fearing that it might be doing harm. Both patients died. When in this third case I again found pneumonia appearing in a case of pericarditis treated with ice, I determined to persevere in my plan of treatment and to push it further. Two more leeches were applied, and a second icebag was placed over the inflamed lung, that over the heart being retained. I should hardly have ventured on this but for the kind willingness of my house-physician, Dr. Gordon (now medical superintendent of the City Fever Hospital, Manchester), who remained by the child's bedside and was able to keep the two icebags in position during the whole night, though the temperature was at times subnormal. When I saw the child next morning the signs of commencing pneumonia had disappeared and recovery was uninterrupted. This case proved to me that with adequate care it is possible to use the ice in pericarditis when the temperature is low. But in the later stages of a pneumonia this is not the case.

The treatment of rheumatic pericarditis by large and frequent doses of sodium salicylate and sodium bicarbonate, with the local application of ice over the heart, is amply justified by its results. Cases treated without ice, and with inefficient medication, are very apt to linger in recovery, and

to result in a permanently dilated and crippled heart. Relapse of rheumatism frequently occurs and ends the case in a year or two. Even apart from a probable relapse, the heart is often so severely injured that symptoms of failure of the circulation develop early. Treatment of the kind here advocated greatly diminishes the tendency to rheumatic relapse; it checks the inflammation, increases the vigour of the muscular fibre, and diminishes the dilatation, thus enormously assisting the forces that make for repair. The practical difference to the patient is often a difference of many years of life.

In the milder cases of rheumatic carditis, those in which there is no evidence of pericarditis, similar treatment (with out the bleeding) may be used with great advantage, though the effect is less striking. Since in every case of rheumatism, acute or subacute, there is dilatation of the left ventricle, as may easily be proved by careful percussion, it is clear that there is always some affection of the cardiac wall, either toxic or inflammatory. The cardiac muscle is weakened, as is proved by the altered first sound, the diffused impulse, and the enfeebled pulse-wave. A murmur indicative of endocarditis may or may not be present. Here, then, as really though not so forcibly as in pericarditis, there is a call for antirheumatic medication and for means of repressing local inflammation. I believe that if this method of treatment were universally adopted in the slighter attacks of rheumatism it would very greatly diminish the number of cases of mitral regurgitation and stenosis in adults, and would prevent an enormous amount of cardiac misery.

REFERENCE.

¹ BRITISH MEDICAL JOURNAL, September 19th, 1903, p. 659.

THE MECHANISM OF RESPIRATION IN PNEUMOTHORAX.

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PNEUMOTHORAX with an external opening is far from an uncommon condition, either as the result of an injury or of surgical interference, such as, for example, in cases of empyema, or tumours of the thoracic wall. If an opening communicates between the pleural cavity and the external air, the respiratory mechanism lacks an all-important factor—the difference of pressure in the lung and pleura is abolished, so that the elastic tension of the lung has free play to cause collapse of that organ. It is, however, a matter of common experience that one pleura may be opened with comparative impunity, and that in these cases the lung does not collapse, but in the majority of cases is forced into contact with the chest wall, and, if not injured itself, will rapidly take on its function once more. The means whereby this is accomplished has been a matter of discussion, and the present state of our knowledge on this point is summarized by West.¹ This author states that there is a pressure in the tubes of the lungs during expiration, amounting to from $1\frac{1}{2}$ to 2 mm. of mercury, while during inspiration there is a negative pressure of about $\frac{1}{2}$ mm. of mercury. He is of opinion that this pressure during expiration has some effect in expanding the lung on the affected side, but is uncertain as to the amount of expansion which is possible under the influence of a pressure of this magnitude. He further notes that although the expansion of the lung may at first be to a considerable extent due to the violent respiratory movements and to the cough which frequently follows the operation, yet that there must be some other explanation, inasmuch as this expansion may take place without any of these violent respiratory movements, and without any cough.

As far as I have been able to ascertain, no satisfactory explanation has been put forward to explain this phenomenon, and it was to attempt to throw some light on this problem that the present series of experiments were undertaken.

There is one factor in respiration which cannot have escaped notice, but which so far has escaped emphasis, and this is the very important resistance to the outflow of the air which can be offered by the vocal cords and fauces generally.

The following are brief notes of an illustrative case, for permission to publish which I am indebted to Mr. Burghard, Surgeon to the Paddington Green Hospital for Children, and to Dr. Alexander Morison, Physician to the hospital.