Meanwhile, let me say that the sudden discharge of pus, bile, hydatid membranes, and the like, by cough and expectoration, may come under our observation at any time as the first symptom or event that compels the patient to seek advice for disease of the liver or other abdominal organ.

I have now related a sufficient number of this series of cases to make the clinical bearing of them obvious; and I may venture to state it in the form of a second clinical rule for your acceptance, as I did the conclusion at which we arrived from a study of my first series. Let it be a rule of practice in every instance of pleurisy and pulmonary disease, or of difficulty in connection with the interpretation of pulmonary symptoms, particularly at the base of the chest, to complete our examination with a careful inquiry into the condition of the stomach, the liver, the intestines, the other abdominal viscera, and the peritoneum itself, remembering that affections of the chest often originate below the diaphragm. I would particularly ask you to observe that the risk of your failing to follow this second rule is greater than the probability of your neglecting the first. As a matter of fact it is not generally observed. Examination of the abdomen is not a routine procedure when the patient's complaint relates to the chest, especially if the patient be a woman. But as I have already said, there is usually another reason for this oversight when we commit it, namely, that we rest content with the diagnosis "pleurisy," "cavity," "hæmoptysis," or the like, which, however elaborate it may sound, is nothing more than a first step towards a working acquaintance with the case. And as the risk of omission is considerable, so also the discredit that may follow it is serious. It is very unfortunate to call by the name of "pleurisy" a grave abdominal disease like perigastric abscess, or tuberculous peritonitis, or cancer of the liver.

Pelvic Disease.

I have spoken so fully on the association of diseases of the abdomen and chest, and raised it to a position of such importance by taking it as the subject of the Hunterian Lecture, that I fear lest I may have gone too far and be responsible for your discovery of it in cases where it does not exist. This is a mistake which is easily made. A good many other conditions simulate very closely that which we have been considering this evening. Let me mention a few of the commonest of them.

In the first place, invasion of the pleura and lung from the abdomen is simulated by secondary affections of the same parts in puerperal septicæmia originating in the pelvis. I need not do more than mention this serious condition, which occasions so much anxiety when it occurs in your midwifery practice. No doubt its pathology varies considerably in different instances, but certainly in cases that I have recently seen the infection reached the chest through the blood stream, not *via* the lymphatics of the peritoneum and diaphragm. This reminds me of a mistake that I once made in the diagnosis of a case of pleurisy following a pelvic operation. A single woman of 35 had a papillomatous mass removed from the neighbourhood of the uterus by combined suprapubic and intravaginal operation. All went well for a few days, but then the temperature rose, vomiting set in, and empyema developed in the right side of the chest. In spite of the fact that there was not a trace of peritonitis, I maintained that the relation of operation and empyema was too obvious to be ignored. But I was wrong. The case recovered after incision and drainage; there was no septicæmia; and the empyema was proved to be influenzal, for it turned out that the patient's sister had come to sit by her bedside straight from that of a friend with influenza.

The second error in diagnosis that might be made by one somewhat over-zealous to discover an instance of the connection which we are discussing, consists in interpreting every patch of dulness and crepitus at the base of the chest as significant of invasion of the lung or pleura. We must not forget that in great enlargements of the liver an area yielding these signs may be due to compression and displacement upwards of the lower lobe of the right lung; and that the same parts may be hypostatically congested and oedematous, if the patient has been confined to bed for some time. Nor are the symptoms always so free from possible fallacies that mistakes may not occur unless scrupulous care be exercised

in the investigation of them. I promised a few minutes ago to return to this point in connection with the case of hydatid of the liver and hæmorrhagic sputum which I described to you. You will remember that I found a hydatid hooklet in the sputum, and the ordinary physical signs of hydatid of the liver. Now the hydatid of the liver, which was a very large one, was treated surgically but unsuccessfully, for the patient died, and then we discovered that the blood stained sputa had not come from the hydatid in the liver at all, but from a second hydatid in the base of the right lung. If I had examined the blood-stained products more carefully I should have been able to determine that they contained neither bile nor hepatic tissue.

Lastly, it is very often difficult, and sometimes impossible, to make a differential diagnosis of the seat of the primary lesion in the abdomen which has invaded the chest, that is to say, whether it is in or around the stomach, the bowel, the liver, the kidney, or one of the other viscera. But this is a part of the subject into which you will not expect me to enter now.

In conclusion, and, as it were, by way of postscript, let me notice two other points of interest indirectly connected with our present subject. The first is a pathological one. We have had before us to-night many instances of the passage of infective materials from the abdomen into the pleura. May not this be the usual route by which the pleura is invaded by the tubercle bacillus? We all know how extremely common tuberculous pleurisy is, and how, as a rule, it precedes the pulmonary disease by months or by years. In the face of what we have been studying this evening, is it not likely that the bacillus reaches the pleura in the lymphatic stream through the diaphragm from the cavity of the peritoneum with its many visceral relations?

The second point is a strictly practical one. Indeed, so thoroughly practical and important is it that this must serve as my excuse for introducing it into a subject to which it does not strictly belong. I refer to the condition of the abdomen in acute disease within the chest, particularly acute bronchitis and acute pneumonia. Distension of the stomach is a very common condition in these diseases, partly produced by free feeding with fluid diet. Intestinal distension is frequently associated with diarrhoea in acute pneumonia. In both cases the diaphragm is driven and kept forcibly upwards against the lungs and heart; respiration and circulation are grievously handicapped, and the patient may perish in consequence of an adventitious embarrassment which is preventable or remediable. Might I venture, then, to submit to you a third rule of practice? Never neglect to examine with particular care the state of the abdomen with respect to distension and pressure in every case of acute pulmonary disease.

THE ACTION OF IODIDES ON THE HEART AND CIRCULATION.

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THE action of iodides on the heart and blood vessels, with their consequent effects on blood pressure, has been the subject of much investigation and discussion owing to the interest arising from their extensive use in aneurysm, arterial sclerosis, and degenerative changes of the cardio-vascular apparatus. When iodides are given for the treatment of these conditions no immediate marked action on the circulation is observable, such, for instance, as takes place on giving nitrites, aconite, or digitalis; and whatever curative effect they may exercise is brought about gradually and slowly, so that only after prolonged administration can any distinct improvement be noticed in the morbid condition. In these cases, moreover, the administration of iodides usually forms only a part of a more or less complex system of treatment, and hence their share in the result as compared with rest, diet, and eliminant measures is very difficult to define. Rosenbach, in his large work on diseases of the heart (Berlin, 1893) expresses the opinion that iodides are of no value in the treatment of aneurysm, and that they are even hurtful in

Rabbit under Ether & Morphine. Sodium Iodide by jugular Vein 18 grains in all.

Tracing 1.—Tracing from carotid artery, no fall of blood pressure.

Rabbit under Ether & Morphine. Potassium Iodide 1 grain by femoral Vein

Tracing 2.—Tracing from carotid artery, transient fall of blood pressure.

arterio-sclerosis, but the great bulk of medical opinion and experience is directly contrary to this. The immediate or visible results of their administration are often disappointing; but if it is considered that the cardio-vascular diseases in which they are given are always of long-standing, and arise from old age, hard work, kidney disease, gout, lead poisoning, syphilis, and other factors, which at this stage are practically incurable and are continuous in their action, it cannot reasonably be expected that any remedial measures can do more than ameliorate or retard their injurious effects on the heart and blood vessels. That iodides exercise a beneficial action in many of these cases seems to be proved by the experience of their almost universal use in practice; that they are not absolutely curative is quite another matter. In any case their action on the circulatory system is a matter of clinical interest.

As a rule when sodium or potassium iodide is given in ordinary doses the pulse remains quite unchanged to the finger both as regards rate and force, hence it is very unlikely that they produce much alteration in the physical conditions of the circulation due either to weakening of the heart or to dilatation of the arterioles. In spite of this, reference to textbooks on pharmacology and therapeutics shows that most authors attribute to the iodides a depressing effect on the circulation and blood pressure, while only a minority hold that there is no adequate proof of this. In special treatises on diseases of the heart and blood vessels their mode of action in aneurysm is frequently explained, but no two authors seem to agree in their explanations. Dr. George Balfour¹ believes their effects to be due to a marked lowering of blood pressure from dilatation of the arterioles with a further possible action on the muscular and fibrous coats of the sac, others hold that iodides tend to favour clotting, either by a direct action on the blood or by weakening the heart beat, while another theory maintains that benefit is seen only in those cases primarily due to a syphilitic taint. Sée and Lapicque assert that it is only the potassium iodide which is useful in heart disease, in asthma, and in arterial disease, and hold that while the potassium stimulates the heart in a manner comparable to digitalis, the iodine exerts a vasodilating action, and that it is the combination of these effects which gives value to the drug. Clinical experience with the sodium iodide is, however, totally opposed to their theory, as the latter salt is found to be equally effective in all these conditions. These various views have been based, very largely at least, on the results of experiments on animals, which, as we shall presently show, are scarcely available for comparison, as they have been carried out under entirely different conditions from those involved in giving iodides to man by the mouth, and often without sufficient reference to the action of the base (sodium, potassium, ammonium, strontium) with which the iodine is combined. Further, it is to be borne in mind that in disease the action of the drug extends over weeks or months, in pharmacological experiments over one or two hours only.

ACTION IN MAN.

We have, therefore, made systematic observations on the blood-tension and pulse-rate of numerous patients who, for one reason or another, were taking potassium or sodium iodide. These observations were made with von Basch's sphygmomanometer and Gärtner's tonometer, instruments which detect and record slight changes in arterial tension. It is not necessary to describe them here in detail, but we may explain that the former is applied to the radial artery, and the amount of pressure required to extinguish pulsation is transmitted through an air-pad and recorded on a graduated dial; and that the latter is applied to the finger, the amount of pressure required to obliterate the blood flow through the digital arteries being transmitted to a column of mercury and recorded on a scale.*

Both instruments are now well known, and are regarded as giving accurate results when carefully used. We satisfied ourselves by observations on patients suffering from pneumonia, cardiac disease, renal disease, advanced phthisis, advanced diabetes, etc., and on others taking such drugs as

nitroglycerine, aconite, and digitalis, that the instruments recorded accurately, and indicated at once changes in the arterial tension and blood pressure. Observations on ourselves and on students attending the class confirmed this, and showed that the instruments could be depended on to record slight changes—that is, changes not detectable by the finger.

We used only the iodides of sodium and potassium, generally in doses of 15 to 30 or 40 grs. per day, a few patients had up to 180 grs., and one man (suffering from spinal sclerosis) was taking 300 grs. Some of these cases had healthy circulatory systems, and were taking iodide for chronic rheumatism, sciatica, facial paralysis, etc.; others suffered from renal or cardiac disease, aneurysm, arterio-sclerosis, bronchitis, etc. One of them had very severe iodism, others had it slightly. Observations were taken regularly during the iodide administration, but in no case did any fall in the blood pressure occur, or any change in the rate or rhythm of the heart. Even the man who suffered so severely from iodism maintained his arterial tension unimpaired, although he said he felt very depressed and ill. In these cases the depression seems to be due partly to the coryza and partly to an action on the nervous system.

The result of the observations is to show that potassium or sodium iodides, given to men by the mouth in therapeutical doses, do not modify the physical conditions of the circulation, and that therefore they neither directly weaken the heart nor dilate the arterioles. The therapeutical effects must be due to some other mode of action, and this is probably true also of some of the rarely-occurring poisonous effects. It is sometimes observed, for instance, that iodides quicken and weaken the pulse, and this has been specially frequent in goitre; sometimes also their administration is followed by emaciation. There is good reason for believing that in neither case is this a direct effect, but is due to increased formation or alteration of the iodine-containing thyroid secretion, which has a powerful action on the circulation and on metabolism.

ACTION ON ANIMALS.

With regard to the action of iodides on the circulation in animals, a considerable amount of experimental work has been done, part of which we have repeated and checked. The total result indicates that iodine or iodides, when given by the mouth, do not alter the dynamical conditions of the circulation. When given intravenously, potassium iodide exerts the marked depressing potassium action on the heart, whereas sodium iodide is practically without this effect.

Tracing 1 is taken from the carotid artery of a rabbit to which 18 grs. of sodium iodide dissolved in water was given by the jugular vein in twenty-one minutes, 3 grs. at a time. There was practically no effect on the heart or blood pressure, except what might be due to irritation of the endocardium by the salt, and which was also produced by similar solutions of sodium chloride. A much larger amount—2.7 grams (about 40 grs.)—given in forty-six minutes had a similar effect. Boehm and Berg,² giving iodine dissolved in sodium iodide to dogs and rabbits intravenously, could discover no action on the circulation at any period of the experiment, a result which has been confirmed by Henrijean and Corin,³ and with sodium iodide alone by Prevost and Binet.⁴

When potassium iodide is administered by a vein the result is very different. All observers are agreed that, given in this way, its action as a cardiac depressant is very marked. Small and carefully-graduated doses, however, cause a transient rise of blood pressure, with slowing of the pulse and increased size of wave. When given by the stomach the depressing action on the blood pressure is only observed after enormous doses, and is never very evident in man. It is an effect of potassium, common to all the potassium salts and seems to be due to a large amount of the potassium salt striking the heart at once and in a certain concentration, for, if the solution be very dilute and injected slowly so that it mixes well with the blood, these results are not seen. Although the blood pressure may sink to *nil*, the heart muscle and nerve ganglia may not be killed, but may recover rapidly as the blood washes the poisonous salt out of the heart. This is seen in Tracing 4. The heart, however, may be poisoned past recovery, and death then occurs in asphyxial convulsions.

* An account of v. Basch's instrument is given in *Zeit. f. klin. Med.*, ii, 1880; *Wien. med. Woch.*, 1883; *Berl. klin. Woch.*, 1887; by S. Fränkel in *Deut. Archiv. f. klin. Med.*, xlvii, 1890; and by Rosen in *Dorpat pharmak. Arbeiten*, vii, 1891. Gärtner's tonometer is described in *Wien. med. Woch.*, 1899.

Tracing 3.—Rabbit under ether and morphine; potassium iodide by vein; 6 grains in all. Tracing from carotid artery, slight rise of blood pressure with fall after a larger dose, slowing of heart, and more ample pulse wave.

Tracing 3.—(Continued).

Rabbit under Ether & Morphine. Potassium Iodide by jugular Vein.

Tracing 4. Showing sudden fall of blood pressure with recovery, and paralysis of heart after a further dose.

Tracing 2 shows the transient depressing effect of a single dose soon recovered from. Tracing 3 shows the rise of blood pressure, also the fall after a larger dose, and the slowing of the heart with a larger pulse wave. Tracing 4 shows a rapid fall and rapid recovery of the blood pressure followed after a further dose by paralysis of the heart.

The difference in action between sodium and potassium iodides when given intravenously is therefore very striking, but it depends entirely upon the base, and is not seen when the drugs are given by the mouth.

Sée and Lapique⁵ have stated that with both sodium and potassium iodide there is a late iodine action consisting in a fall of blood pressure from dilatation of the arterioles. This we have never seen, and its occurrence is denied by other observers also. Bogolepoff⁶ is frequently quoted also as an authority for the statement that potassium iodide dilates the small vessels, but his conclusions on this special point are largely based on microscopical examination of the capillaries of the frog, a method of no value for determining the question. As a matter of fact, the perfusion of a dilute sodium or potassium iodide solution through the blood vessels of a frog does not cause them either to contract or dilate; it has as little action as normal sodium chloride solution has.

We also made a large number of experiments on the heart of the frog isolated and attached to Williams's apparatus. When iodine was added to the circulating fluid (1 in 3,300 parts, 1 in 1,000, etc.) it acted sometimes rapidly and sometimes slowly as a muscle poison, the beat gradually becoming smaller and the heart stopping in diastole. Sodium iodide was also rather irregular in its rate of action, but the ultimate result was to weaken the heart muscle and finally to poison it. Sometimes peristaltic contractions passed over the heart and there was marked systole such as occurs in the frog heart with digitalis. With weak solutions of potassium iodide (1 in 1,000 or 2,000) the digitalis-like action was very evident and is due to the potassium. The beats become much more ample, and the systole and diastole very complete. Stronger solutions (1 in 400) killed the heart muscle in a few minutes. With ammonium iodide the heart was slowly killed, very much as with the sodium salt.

These experiments on frogs do not, however, throw much light on the action of iodides on the heart and circulation in man and mammals. As we have already shown,⁷ iodine and iodides exercise a very poisonous effect on the muscles of the frog, no trace of which can be observed in mammals, and the action on the frog's heart is probably just a phase of this. When large doses of sodium iodide or sodium chloride (40 to 60 grs. or more) are given to a rabbit by a vein, the animal shows no symptoms for some hours and then dies of oedema of the lungs and pleuritic effusion. It is apparently a saline action independent of the iodine. If potassium iodide be given in the same way a few grains produce instant death, this being a potassium action on the heart. Potassium salts, whether iodide, bicarbonate, acetate, or any other, when given by the mouth in ordinary quantities, reach the heart so diluted by the blood that they usually do not show even a trace of this depressing effect.

We may conclude, therefore, that sodium or potassium iodide when given to man by the stomach in ordinary doses has no depressing effect on the action of the heart or on the blood pressure in the arteries.

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THREE CASES OF RUPTURE OF THE LEFT VENTRICLE.

BY

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THE following are three examples of that very rare pathological condition—rupture of the heart. The first two are of particular interest from the similar features, both clinical and pathological, that they present: in each case there was abso-

lutely no history of any exciting cause, such as excitement or sudden exertion, the patient being in bed in one case, and quietly sitting on a seat in the other. In all three cases the lesion was situated in the wall of the left ventricle.

Case 1.*

J. B., male, aged 48, was admitted into the Dorset County Asylum in 1861 suffering from mania with delusions. He was then in good bodily health, but no previous history was ascertained. Up to the time of his last illness there was nothing of special interest in the history of this case. Mentally, he became demented.

On September 30th, 1901, he appeared to be rather out of sorts, and was put to bed. On examination no marked symptoms presented themselves.

At 8.10 A.M. on October 2nd he had taken about a pint of milk, and got out of bed for about three minutes; and at 9.30 A.M. he appeared in his usual condition, and told the attendant that he was "all right." At 10.30 the attendant noticed his head fell over on the pillow, and he appeared to be unable to draw breath. The medical officer was at once called, and the patient was found to be moribund, and he died two minutes afterwards.

Fig. 1.—Drawing from original specimen by Mr. G. A. Rorie.

Necropsy.—This was made on the next day. On opening the pericardium the cavity was found to be filled with dark-coloured clot covering the anterior surface of the left ventricle, and there was also a considerable quantity of bloody fluid present. The heart was not adherent to the pericardium. The heart was found to be somewhat dilated, was soft and flabby to the touch, and there was a considerable amount of fat on its surface.

In the anterior wall of the left ventricle was a tear running in the line of the muscle fibres. It was 1 inch in length, and was situated about 1 inch from the apex and $1\frac{1}{2}$ inch transversely from the interventricular septum; it communicated directly with the cavity of the left ventricle. There was no evidence of localised disease at the point of rupture. On microscopical examination the heart muscle was found to be in an advanced state of fatty degeneration, and also showed fatty infiltration. Beyond this condition of the heart muscle there was no actual disease about the heart. As regards the other organs, the only points of interest were some softening of the brain and primary cancer of the gall bladder and liver.

Case 2.

The second case—for the notes of which we are indebted to Dr. Rorie, Dundee—was a female, aged 50, when admitted into the Dundee Royal Lunatic Asylum on June 23rd, 1893, suffering from alcoholic dementia. On admission she was in fair bodily health and condition, and her various systems were healthy, beyond the fact that her pulse was quick and rather weak; her eyes showed signs of old keratitis; she was very deaf, and mentally she was in a condition of dementia.

Her previous history showed that she had been addicted to alcoholic excess for some years, and this had been more marked recently. She had had a family of eight—two sons and six daughters—one son being epileptic, and two children were stillborn.

As regards the progress of this case there is nothing particularly of interest; her bodily health improved somewhat, but mentally she remained demented and degraded in habits.

On May 18th, 1901, she complained of feeling sick at 5 A.M., but got up at the usual hour, went about the daily routine as usual, and took a good breakfast. At 11 A.M., while sitting quietly on a seat outside, she fell off

* The specimen was exhibited by Dr. P. W. MacDonald at the autumn meeting of the Dorset and West Hants Branch of the British Medical Association, October 17th.