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.

the seat "as if in a fit," was at once carried inside and laid on a couch. She was at once seen by the doctor, and found to be dead.

Necropsy.—This was performed next day. On opening the pericardium a large amount of clot was found lying in the anterior surface of the heart, and distending the pericardium. The heart was fatty as a whole, and showed two medium-sized milk spots on the left ventricle. At a distance of 1 inch from the apex, on the anterior aspect of the left ventricle, was an area reddish-purple in colour from effused blood, in the centre of which was a small irregular tear about $\frac{1}{4}$ inch in length. On inserting a probe it was found to lead upwards and inwards into the cavity of the left ventricle, about $\frac{3}{4}$ inch from the interventricular septum. Otherwise there was nothing of note, except that the whole heart showed marked fatty change; it weighed 9 ozs. As regards the other organs, there was little of interest; the lungs were emphysematous, the liver and kidneys were somewhat fatty, and the brain showed signs of degeneration.

Fig. 2.—Drawing from a photograph by Mr. G. A. Rorie.

Case 3.

J. S., aged 79, was admitted to the Dorset County Asylum in October, 1900, suffering from senile mania. The previous history shows nothing of importance, and he had been in fair bodily health for his age up to the time of his death.

On going to bed on the evening of November 8th, 1901, he had a quarrel with another patient, in the course of which they both rolled back on his bed. On the attendant separating them, the patient got up and walked into another dormitory, discussing the subject of the quarrel with the attendant. He then sat down in bed, asked the attendant not to leave him, and fell back in bed unconscious. He was at once seen by the medical officer, and found to be unconscious, slightly cyanosed about the lips, and pulseless; after one or two shallow respirations he died.

Necropsy.—A post-mortem examination was performed next day. On opening the pericardium it was found full of bloody fluid, and covering the entire surface of the heart was a thick layer of dark clot. On removing this, a small fissure was found on the posterior aspect of the heart. It was exactly $\frac{1}{2}$ inch in length, 1 inch from the auriculo-ventricular septum, 1 inch from the border of the pulmonary artery, and followed the direction of the muscle fibre. It communicated directly with the cavity of the left ventricle. The heart as a whole was very fatty; there was a milk spot on apex of left ventricle; the muscle was soft and friable; the mitral valve was much thickened, and there was some calcification of the cusps of the aortic valve, which, however, was competent. Otherwise there was no other naked-eye pathological change noticeable. The other organs of the body were healthy.

THE German Balneological Congress will hold its twenty-third annual meeting at Stuttgart, March 8th to 12th, 1902. All communications should be addressed to the General Secretary, Geh. Sanitätsrath Dr. Brock, Melchiorstrasse, 18, Berlin, S.O.

ATHEROMATOUS ULCERATION OF THE HEART: PERFORATION: SUDDEN DEATH.

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As ulcerative perforation of the heart is extremely rare, the following case, which came under my care recently, will probably prove of interest. The notes were taken by Dr. Clark, the house-physician.

C. H., aged 62, a manufacturer's agent, was admitted on September 16th into the Hertford British Hospital, Paris, suffering from "pain in the chest and dyspnoea."

History.—He had not been feeling well for five days, and had to lie up on account of pain in the chest running through to the back, the pain being more intense on the left side. As this had increased, notwithstanding the medical advice obtained, he had come to the hospital. He had seen better days, but for the last twelve years had led a very anxious life. There was no history of tubercle or syphilis. About a year ago, he had an acute attack of pleurisy and nearly succumbed; his urine then contained albumen, but it disappeared afterwards.

Condition on Admission.—Patient was of medium height, florid, short neck, and stout. Temperature 99.6°; pulse 104; respirations 31. Breathed with difficulty, and lay in a semi-recumbent position. The lungs were emphysematous, with signs of pleurisy over the lower third of back and side of left lung, but none over right lung. The heart's dulness was not made out owing to emphysema of the lungs. The impulse was in the fifth space and in the nipple line. The heart sounds were distant, the first sound prolonged at apex, but no bruits were heard anywhere. The pulse at the wrist was quite regular, strong, and compressible. There was pain on palpation of epigastrium. The liver and spleen could not be felt on account of fat in the abdominal wall. Percussion was not attempted, as it distressed the patient. Examination of the kidneys was for the same reason omitted. The urine was acid, specific gravity 1030, of high colour, and contained 0.5 parts per 1,000 of albumen.

Treatment and Progress.—Linseed poultices were applied over the front and back of the chest. A cathartic pill and a diuretic mixture, milk, and barley water were given. The next day (in the morning) the patient was better, the pulse normal, and the pain local. In the afternoon, at about three o'clock, pain returned, urine was passed in larger quantity, he perspired profusely, looked very anxious, and said he felt he was dying. Four hours later, whilst the nurse was changing the poultices, the patient suddenly fell back, had twitchings of the hands and face, and died.

Necropsy (17 hours after).—The dura mater and pia mater were thickened, with atheromatous patches in several places; the sinuses were gorged with blood. The arteries were atheromatous. No clots or effusion of blood were found in the brain substance. The right lung was emphysematous; the left showed old adhesions to the chest wall; the lower lobe was collapsed (*post mortem*), owing to pressure of the large pericardial effusion. The heart was hypertrophied and dilated, with much fat on its outside. Its muscle was fairly good. The coronary arteries were atheromatous. There was a perforation as big as a small crowquill on the posterior wall of the left ventricle, or about midway from the upper and the lower extremities, and about half an inch from the interventricular septum. This perforation was in the centre of an ecchymosed patch. The pericardial sac, the front of which was adherent to the chest wall, contained about 8 or 10 ozs. of blood clot. On opening the left ventricle of the heart it was found that the perforation corresponded with a ragged, funnel-shaped ulcer, the size of a shilling, which had given way at its apex; and about an inch lower down there was another smaller ulcer which extended as deeply as the visceral layer of the pericardium. The branches of the coronary artery near these ulcers were blocked for about $\frac{1}{2}$ inch by thrombi of an atheromatous nature. Evidently these had been the cause of the ulceration. The mitral valves showed old vegetations. The aortic were competent but contained small particles of chalky material. The pulmonary and tricuspid valves were not diseased. The aorta was dilated and very atheromatous a short distance from the heart. The liver, spleen, and intestines were normal. The kidneys were granular, with small retention cysts. Their substance was rather hard; the cortex was slightly diminished; the suprarenal capsules normal.

ACUTE RHEUMATISM: HYPERPYREXIA: POST-FEBRILE MANIA: RECOVERY.

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THE condition of hyperpyrexia in acute rheumatism is so uncommon that I have thought it worth while reporting the following case:

W. W., aged 32, married, a gardener, very dark complexion, nervous temperament, intelligence under average.

History.—Family history could not be ascertained. His previous health was fairly good; he had suffered from colds and indigestion, but had had no acute illnesses. During the early three months of the year he had felt weak and out of sorts, had had little appetite, and his food had disagreed with him, but prior to the onset of his present illness he had no rheumatic symptoms. He first felt pain in the right wrist, which was swollen, on April 6th. He thought he might have sprained it at his work. Next day he went to work, but by afternoon he had to knock off, as his right knee became affected; by night both knee-joints were so painful that he got no sleep.