A Post-Graduate Lecture ON CARDIOLYSIS.

BY
SIR ROBERT M. SIMON, M.D.,

SENIOR PHYSICIAN TO THE GENERAL HOSPITAL, BIRMINGHAM.

In 1902 Brauer suggested that, in cases in which it was suspected that the free action of the heart was hampered by adhesions of the pericardium to parts outside it, it would benefit the patient to remove some of the bony and cartilaginous framework of the thorax. He supposed that the precordial part of the chest wall would thereby become more flexible and oppose less resistance to cardiac expansion. It is probable, however, that the relief to the heart thus given may lie less in the softening of the wall against which it beats than in the removal of tight and tough adhesions to the fixed points like the ribs, and that relief may be expected from the removal of these adhesions.

Three of his cases were operated on, and 17 in all were recorded up to 1908. No death occurred as the result of operation, and benefit resulted in each case.

In 1908, Poynton and Trotter brought the case of a patient on whom the operation had been performed before the Royal Society of Medicine, and were of opinion that the proceeding had been justified by the improvement in the boy's condition. It must be remembered that when the operation has been performed, it has been done to relieve symptoms which have resisted all other methods of treatment, and often when, as in my own case, the patient has appeared to be on the very threshold of death.

Another case was referred to at this meeting in which death occurred three months after the operation owing to a fresh attack of pericarditis. From a surgical point of view the discussion narrowed itself down to the point whether or no the whole periosteum of the ribs should be removed—the authors of the paper concluding that the anterior periosteum only should be taken away, reossification under these circumstances not being likely to occur. Accidents to the left pleura, which comes close to the sternum, would in these circumstances be less likely to happen.

In my own case the left pleura was opened twice without any harmful result beyond a trifling pneumothorax, and I think it extremely probable that pneumothorax to any considerable extent is unlikely to occur, seeing that in every case in which cardiolysis is to be useful, the pleura and pericardium will be found to be united in dense adhesions which extend very much further to the left and right than the necessary incisions.

The pericardium is normally adherent to the central tendon of the diaphragm as well as to the sternum, and in a long-standing case of adherent pericarditis the adhesions between the two may be much more extensive than in a normal state. Whether it would be possible without danger to life to cut through these adhesions is a matter for consideration, as I do not doubt that the enlargement of liberty for a heart, if freed from its adhesions to bone, would be enormously increased by diminishing its area of anchorage to the diaphragm.

The following is in brief the record of the condition of my patient before admission and during his stay in hospital:

H. S., an office boy, aged 15, had enjoyed quite good health until the age of 12 years, when he had pleurisy on the left side and was in bed for three weeks. After he got better he felt quite well, but was told not to run, as his heart was affected. He had had growing pains in the legs since the age of 11, but never rheumatic fever. He had not suffered from sore throats. The family history was negative.

Ever since the pleurisy he had noticed that he had been short of breath on exertion. At first this was very slight and he took a good deal of exercise, especially swimming, but he had to give this up on account of shortness of breath. His employment in an office had involved walking upstairs. For a long time he had been quite unable to run, and recently the office stairs have tried him very much. Since February 17th, 1912, his legs and feet had been swollen. He went to bed on the 14th and his abdomen soon became very swollen. He was tapped and 9 pints of fluid were removed.

Condition on Admission.

On May 14th, 1912, when he was examined, he was pale, the cheeks and lips were cyanosed, and the conjunctivae glistening and slightly oedematous. He was propped up in bed and had marked dyspnoea, which was increased by talking.

The cardiac impulse was forcible and heaving; it was not well localized, but was best felt in the seventh and eighth left interspaces in the mid-axillary line. There was bulging of the whole precordial region, the ribs being pushed out with each systole, while below and laterally there was systolic indrawing. The percussion area was extremely wide.

The first sound at the apex was loud, long, low-pitched, and booming; it was not followed by any definite murmur. The second sound was replaced by a distinct harsh diastolic murmur. There was a suggestion of a presystolic murmur. The first sound in the pulmonary area was followed immediately by a soft systolic murmur; the second sound was loud and reduplicated, and there was a diastolic murmur. There were double murmurs in the aortic and tricuspid areas.

The pulse was small, weak, of low tension, 112, regular.

There was some oedema of lungs at both bases and a little generalized bronchitis.

The liver was one fingerbreadth above the umbilicus and tender; marked ascites was present. The fingers were slightly clubbed and reddened at the extremities; the legs were oedematous.

On May 17th 14 ounces of fluid were removed from the right pleura.

On May 29th the quantity of urine was very small. The pulse had been continuously above 100 since admission, and feeble; 7½ pints of fluid were removed from the abdomen.

On May 30th he seemed slightly better. On June 2nd the cardiac impulse was more localized and forcible under the seventh left rib, 5½ in. from the mid line.

Operation.

On June 17th the patient was anaesthetized with a mixture of nitrous oxide and oxygen, atropine and morphine having been injected subcutaneously first. Under the anaesthetic he was very bad, and artificial respiration was performed for a short time. Mr. Baring removed the sixth, seventh, and eighth costal cartilages and ribs on the left side for about 4 inches from the sternal margin, through a wound made by turning up a large flap of skin. During the operation the pleura was opened in two places and a pneumothorax produced. The pleura appeared healthy, but the pericardium was very adherent to the anterior thoracic wall. The operation occupied about twenty minutes. After the operation the boy was remarkably well and had comparatively little pain.

On June 21st his pulse-rate, which had almost always been above 100, suddenly fell to 60. He had been having "Guy's pills," one three times a day. On June 29th no diastolic murmur could be heard anywhere. The boy seemed more comfortable than he had been before, and his heart was smaller.

On July 2nd a tracing showed a totally irregular pulse without any trace of auricular systole.

Since July 12th the boy has been up and about. Although his pulse has been continuously irregular he is comfortable and much less cyanosed than formerly. His breathlessness has vanished and also the clubbing and redness of the finger tips. He has no oedema, and is well able to walk up and down stairs at present.

It will be seen that there had been no definite history of rheumatic fever, though the so-called growing pains were probably of rheumatic origin, as, when he recovered from the attack of pleurisy at the age of 12, he was warned not to run because his heart was affected.

From the time of his pleurisy he was short of breath, but did a good deal of swimming until dyspnoea made him give it up. He remained pretty well until February of this year, when he had to give up work as an office boy because he could not climb the stairs to the office, and took to his bed on February 14th. His feet and legs were swollen, and he soon developed ascites which necessitated tapping and the removal of nine pints of fluid. I saw him in consultation with Dr. Hadley of the General Dispensary, and at the request of his friends admitted him to the General Hospital about the second week in May.

Prior to that date he had not shown any sign of improvement under any and every treatment, and I warned his friends that he was not likely to do well. They quite understood this, but wanted him to have the better nursing and comforts of a hospital. They agreed again when I proposed operation a few weeks later, after having failed to do more than temporary and very transient good by tapping the abdomen and pleura, and by the use of cardiac tonics.

Despite the various bruits that were noted by my house-physician, Dr. Wilkinson, I could never satisfy myself that there were valvular lesions enough to account for all of them, and became more and more impressed by the belief that there were not only adhesions between the fibrous bag and the serous sac of the pericardium, but that the

fibrous bag was adherent to the front of the thorax, the diaphragm, and the pleura.

In consequence of these external pericardial adhesions I imagined that proper muscular contractions of the heart wall had become impossible, and that the bruits might be due to distortion of the cavities and their openings. The boy was distressingly short of breath, very miserable and in great discomfort, and it seemed clear that death was imminent. Under these circumstances I felt that the separation of the external adhesions would at least give the heart some chance of proper contraction, would increase the comfort of the patient, though the risk of his dying under anaesthesia was recognized. Mr. Barling completed the operation in twenty minutes, and from the moment of recovery from anaesthesia the boy became more comfortable than he had been for months.

The patient has made an uninterrupted recovery in so far as breathlessness, distress, and oedema of legs or feet or abdomen are concerned. The liver has become normal in size, the enlargement of the spleen has disappeared, and, most important of all, the diaphragmatic movements, though still limited, have become more extensive, while the dilatation of the heart has been much reduced. All the bruits, with the exception of systolic mitral, have disappeared, and the boy can walk, sleep, and enjoy life.

Three days after the operation the pulse, which for months had never been under 100, dropped to 56. He was taking digitalis at the time, and, though this was very soon stopped, it has not once reached 100 again, but is usually between 70 and 80 and is fairly regular.

The pulse tracings indicated auricular fibrillation, and it is pretty certain that there is, as was to be expected, a considerable amount of degeneration of the myocardium, so that it is improbable that he will ever be fit for much muscular effort; but he is alive and comfortable, and this result is solely due to the operation.

The selection of a suitable case for this treatment

involves the greatest care and prolonged observation. It is difficult enough to diagnose an adherent pericardium when the fibrous bag is united inseparably to the serous

covering of the heart muscle, but to feel sure that in addition there are external adhesions to the chest wall and to the diaphragm is a most serious problem. It is a difficulty that must be faced, however, or the operation will be discredited and possibly some lives sacrificed. Probably the best way to succeed in making a diagnosis is to determine if there is any way of explaining the increased size of the heart as due to any other cause than old pericarditis.

These cases occur almost entirely in young people at the time of development, in whom it has been known that they have been suffering from heart disease of some standing. Generally speaking, disease of the aortic valves, Bright's disease, and adherent pericarditis are the only causes of hypertrophy of the heart in children, and it is fairly easy to satisfy oneself as to the existence of either of the first two causes, so, by methods of exclusion,

arriving at the diagnosis of old pericarditis. Chronic myocardial degeneration will cause dilatation of the heart and an increase in the area of precordial dullness, but it is rare in children; it may occur, but is seldom rapid in its course. Adhesions of the pericardial membranes must in course of time result in myocardial changes, but, as already stated, they are slow in development, and simple pericardial adhesions may, and often do, exist for many years without imperilling life; so, if in a youth we find a fairly sudden and rapid failure of heart power, and are able to exclude disease of the aortic valves as accounting for the hypertrophy, we are forced to consider the question of external adhesions.

We are helped materially by two very important observations—the nature of the impulse and the movements of the dia-

phragm. The latter can be observed in two ways—by radiography and by the movements of the abdominal wall. In inspiration the diaphragm descends, pushing

Fig. 1.—Diagram to show the openings in the pericardium after the removal of the heart. S.V.C., Extrapericardial part of superior vena cava; S.V.C., intrapericardial part of same; P.V.R., P.V.R., upper and lower right pulmonary veins; I.V.C., intrapericardial part of inferior vena cava; I.V.C., extrapericardial part of same above diaphragm; A., aorta; P.V.L., pulmonary artery; P.V.L. and P.V.L., upper and lower pulmonary veins of left side; D., diaphragm.

Fig. 2.—Section of heart in situ to show inferior vena cava and its relations to pericardium and diaphragm.

before it the abdominal wall; if this movement does not occur, the inference is that the diaphragmatic contraction does not take place, because it is adherent to the pericardium, which maintains an almost constant position, owing to its relations to the root of the lungs and the aorta and pulmonary artery, though its junction to the central tendon of the diaphragm causes it to descend a little when the diaphragm descends.

The impulse in these cases is often very diffused and the apex of the heart can generally be felt in the same position, not moving if the patient changes his position and lies on either side, the whole pericardium being anchored and fixed by adhesions. The limitation of diaphragmatic movement has a very important bearing on the liver. Physiologically it is compressed by the descent of the diaphragm, and in its turn presses on the vena cava, so that the blood from the hepatic veins is pumped up through the inferior vena cava into the right auricle. If the diaphragm does not act, this free passage of blood does not take place; it is dammed up in the liver, which becomes swollen, and, as in the case of H. S., may be felt as low down as the umbilicus. If, therefore, in a patient who has been known or suspected to have adherent pericarditis we find such enlargement, and this is not due to cirrhosis, we may feel pretty confident that the diaphragm is adherent to the pericardium, especially if the diaphragm moves less than normally, and the anterior wall of the abdomen does not protrude with inspiration.

The vena cava passes between the right and central lobes of the central tendon of the diaphragm, and it is more than likely that its shape may be altered and its lumen diminished by an increase of the connective tissue of the tendon, due to inflammatory proliferation.

This leads me to express the hope that it may be possible, by means of removal of this pressure on the vena cava, to still further increase the relief that the present operation of cardiomyolysis has rendered. It would, of course, necessitate a second operation on the right side, and it may indeed be impossible, as the vena cava is deep in the thorax and has very intimate relations with the phrenic nerve. Mr. Billington, resident pathologist at the General Hospital, has been kind enough to make a number of investigations for me on the relations of the inferior vena cava in the thorax, and finds that there is a very appreciable length of the vein, an inch or more, which is to be seen in the thorax between the points of piercing the diaphragm and entering the heart.

All the older textbooks speak of the vein as passing through the diaphragm and entering the pericardium immediately. This is certainly not the case, as the vein can be distinctly seen as stated for $\frac{3}{4}$ in. before it pierces the pericardium; the rest of it, comprising about $\frac{1}{2}$ in., is within the pericardium and partially covered by the serous sac. The later textbooks speak of and illustrate this fact.

It is just this first part which may be affected by adhesions of the right pleura, which are very apt to occur in cases of anterior mediastinitis secondary to pleurisy or pericarditis. It is to the possibility of dealing with this condition that I venture to draw the attention of surgeons. The vena cava, when it leaves the diaphragm, is extra-pericardial and is covered by fibrous tissue; what this fibrous covering is seems doubtful. It may be a derivative from the diaphragm or from the fibrous bag of the pericardium, or be independent of both. It is not covered by the pericardial serous sac until about $\frac{1}{2}$ in. before it enters the right auricle. This $\frac{3}{4}$ in. of vena cava, which has as yet no connexion with the serous sac of the pericardium, is therefore very likely to be affected by any proliferation of connective tissue, such as occurs in mediastinitis; but it is equally obvious that adhesions of the two layers of the serous sac—namely, that overlying the heart muscle and that lining the fibrous bag of the pericardium—cannot affect this part at all.

It is possible, therefore, to understand why internal adhesions of the pericardium may occur without inducing any signs of pressure on the cava, such as obstruction to the entrance of the blood into the right auricle, and consequent increase in size of the liver. When such signs occur it would follow that we may assume that external adhesions have been formed. This interpretation, if correct, would explain why sometimes chronic pericarditis may exist for years without producing any symptoms,

until gradually those of progressive myocardial degeneration assert themselves, and, on the other hand, why very rapid signs of heart failure without general valvular lesions may probably be found to be dependent on external pericardial adhesions.

It is in this latter type of case that cardiomyolysis may be expected to prove useful.

The anatomical conditions of the vena cava superior as regards its relation to the pericardium are exactly like those of the vena cava inferior, but the symptoms associated with pressure upon it will be those of delay of the venous circulation in the face and neck.

Cyanosis, which was so marked in the case of H. S., disappeared at once after the operation.

No attempt was made in this case to look for adhesions to the diaphragm or the veins, so it is clear that the beneficial result was due to the increased freedom of contraction from removal of the ribs, and by separation of the adhesions between them and the pericardium.

AN INTRACELLULAR PARASITE DEVELOPING INTO SPIROCHAETES,

FOUND BY THE JELLY METHOD OF IN VITRO STAINING IN SYPHILITIC LESIONS AND IN THE CIRCULATING BLOOD DURING THE SECONDARY STAGES OF THE DISEASE.*

(With Special Plate.)

By EDWARD HALFORD ROSS,

(THE JOHN HOWARD MCFADDEN RESEARCHES) THE LISTER INSTITUTE.

1. An Intracellular Parasite of Guinea-pigs developing into Spirochaetes.

By the jelly method of *in vitro* staining,^{1,2} certain peculiar inclusions found embedded within the mononuclear leucocytes of the blood of guinea-pigs and known as Kurloff's bodies were shown to be parasites.¹⁰ These same bodies, if examined by the usual methods involving drying and fixation, merely appear as opaque semi-stained "splodges," and therefore can be easily passed over as artefacts. Hence their true nature was not recognized until the jelly method was employed. The method enabled the development of this parasite, which was named *Lymphocytozoon cobayae*, to be demonstrated. It showed how the chromatin within the inclusion becomes formed into spirochaete-like bodies, and how, after the inclusion has burst, the spirochaetes swim freely in the blood. They can then be seen readily by the dark-ground illuminator as motile filaments swimming in the blood of the guinea-pigs. In addition to this, amoeboid forms were also observed. In the description of this parasite, the suggestion that these spirochaete forms might be microgametes (male element or spermatozoon) was quoted⁷; and the observations were made that the parasite produces anaemia, and that many of the infected guinea-pigs—a large percentage of guinea-pigs bought in England are infected—exhibited single or multiple white patches or tumours within the substance of the liver and spleen.

2. The Histology of the White Liver Tumours of Guinea-pigs.

Microscopic sections of these gumma-like tumours of the liver and spleen of guinea-pigs were then furnished me,³ and these showed that the tumours contained masses of mononuclear cells, the majority of which exhibited the cell inclusions known as Kurloff's bodies.

3. The History of the Parasite of Guinea-pigs.

Kurloff⁸ thought that the cell-inclusions were vacuoles; another⁴ suggested "Secretstoffe"; while again others^{5,6} claimed their parasitic nature. More recently it has been maintained⁹ that they belong to the Chlamydozoa or Cytoryctes. The supposition of their parasitic nature, however, led to conjectures as to the mode of transmission, and food, ectoparasites, and contact have been suspected. But I have already mentioned that by means of the jelly method these Kurloff bodies have now been proved to be living parasites; and observations point to the transmission of these parasites from guinea-

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