the remarkable results of experimental ligature of these vessels in animals than to extended clinical observation. Sir J. E. Erichsen was one of the first to ligature the coronary arteries in animals (*The London Medical Gazette*, 1842, p. 561) taking for his text a speculation of Dr. Marshall Hall's that interruption of the coronary circulation would rapidly cause death. Since then the experiment has been repeated by a number of observers.⁶ The results obtained were usually disturbance of the rhythm, slowing and finally standstill of the heart. They were, however, far from constant, differences being recorded in the time which elapsed before the heart came to a standstill, even when the same species of animal was employed, and as might be expected different animals reacted differently.

Panum⁷ experimentally injected tallow into the coronary arteries, and noticed that the left ventricle ceased to beat 75 minutes and the right ventricle 90 minutes from the time of the injection. According to Cohnheim (*Lectures on General Pathology*, vol. i, p. 35, New Sydenham Society) ligature of any large branch of either coronary artery in a dog produces, after a quiescent interval of 90 seconds, irregularity and slowing of the heart; this is followed not later than two minutes from the ligature of the vessel by sudden stoppage of both ventricles in diastole. After this the heart cannot be roused to renewed contraction. This sudden stoppage he regarded as probably due to the effect produced on the cardiac ganglia by the occlusion of the vessel, but he admits that the distribution of the ganglion cells within the muscular mass of the organ makes it impossible to determine with certainty whether the arterial anæmia affects the nervous or muscular elements. Apart from the view that the cardiac failure is due to failure of nutrition of the cardiac ganglia or muscular tissue, there is the possibility that it is not the mere absence of blood and nutritive material, but the presence of toxic substances generated by the active tissue metabolism which is responsible for the fatal termination. Under normal conditions such toxic bodies are carried away as soon as manufactured, but with arrest of the circulation they might accumulate *in situ* and paralyse the neuro-muscular apparatus of the heart.

The rapid and sudden failure certainly suggests that the cause, whether it be anæmia or a poison, affects primarily the nervous regulation rather than the musculature of the heart, inasmuch as the latter is less sensitive to external influences and has a reserve power which lasts longer than that of the former.

It is noteworthy that while experimental ligature of the coronary arteries in animals or embolism in the human subject may rapidly cause death, gradual occlusion of the vessels as in endarteritis need not have this effect. The gradual diminution in the supply of blood when the trunks or orifices of both coronaries are obstructed would, it may be supposed, allow of some degree of acclimatisation to the altered conditions taking place. When, as is usually the case, one coronary artery only is markedly affected by endarteritis, compensation may be effected by a dilatation of the anastomoses, which Wickham Legg and S. West have shown to take place normally between the two vessels.

A difference between Cohnheim's experiments and this case is the duration of life after the occlusion of the vessel. In Cohnheim's observations on dogs death occurred in two minutes; in this case about ten minutes elapsed. That this is not a point of any great importance or difficulty is shown by the fact that experimentally the results are not quite the same in rabbits as in dogs. If there be a difference between animals in the way they react to experimental ligature or compression of the coronary arteries, a similar variation may well be expected between animals and man.

It does not seem certain what interval elapses between embolism and death in man. Osler (*Principles and Practice of Medicine*, edition ii, p. 676) says, "sudden death not infrequently follows blocking [he is speaking of embolism as well as thrombosis] of one of the branches of the coronary artery and the production of anæmic necrosis." But anæmic necrosis would take some little time to develop, and although in such cases death might be quite sudden it would not be an immediate result of coronary embolism.

⁶ For a summary, vide Porter, on "Ligation of the Coronary Arteries," *Journal of Pathology*, vol. xv, p. 121.

⁷ Virchow's *Archiv.* 1862. Bd. xxv. S. 312.

In a case of embolism of the coronary artery referred to by Dr. Cutler (*Pepper's System of Practical Medicine*, vol. ii, p. 831) death occurred 20 hours after the commencement of symptoms thought to be due to the embolus. It thus differed considerably from my case, in which death followed the onset of symptoms in ten minutes, and where from the absence of any anæmic necrosis in the distribution of the blocked vessel there was no reason to believe that the interval between embolism and death was longer than it appeared to be from clinical evidence.

THE LEFT AURICLE IN MITRAL STENOSIS: HYPERTROPHY AND DILATATION.

By D. W. SAMWAYS, M.D., D.Sc., M.A., B.C.,
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PROFESSOR OSLER, in his *Principles and Practice of Medicine*, refers to the changes which take place in the left auricle in cases of mitral stenosis in these words:

"The left auricle discharges its blood with greater difficulty, and in consequence dilates, and its walls reach three or four times their normal thickness." "Eventually the tension is increased in the pulmonary circulation owing to impeded outflow from the veins. To overcome this the right ventricle undergoes dilatation and hypertrophy, and upon this chamber falls the work of equalising the circulation."

A somewhat similar statement occurs in Dr. Fagge's *Medicine*,¹ where one reads, "The effect of mitral stenosis on the heart will naturally be to produce dilatation and hypertrophy of the left auricle."

These statements I believe to be misleading and erroneous, especially as given by Professor Osler, who seems to regard dilatation and hypertrophy as being both useful factors in maintaining the circulation. In the first place, it is probably an error to suppose that dilatation of the left auricle exists to any marked extent in compensated mitral stenosis, and in the second place, if it exists, it is a mechanical error to suppose that such dilatation aids the cardiac chamber to overcome resistance in front. The characteristic alteration in the left auricle in mitral stenosis is not dilatation but hypertrophy, as is proved by the following statistics.

During the four years 1888-91 inclusive there were 70 cases of mitral stenosis examined and reported on in the *post-mortem* room of Guy's Hospital. Hypertrophy of the left auricle is stated to have occurred in 36 of these cases. The 70 cases are divisible into two classes almost numerically equal: those in which the stenosis was severe, one finger (55 millimetres) or less, and those in which the stenosis was less severe; 36 cases belonged to the former class and 34 to the latter.

In 15 of the 36 cases of severe stenosis, the left auricle was hypertrophied without dilatation, the hypertrophy having generally been considerable. In 11 other cases the auricle was both dilated and hypertrophied. In only 3 cases, and in those the orifice measured 5 centimetres at least, was the auricle dilated without being hypertrophied, thus showing a marked contrast to the tendency to hypertrophy. No note was made of the condition of the remaining 7 cases, except in one instance where the auricle was somewhat vaguely stated to have been "a little large."

There was, therefore, hypertrophy of the left auricle observed in 26 of the 36 cases of severe mitral stenosis, dilatation existed only in 14 cases, and was associated with hypertrophy in 11 of these cases.

It must not be forgotten that in *post-mortem* records we have the register, not merely of the cardiac condition in compensated mitral stenosis, but also of the changes associated with the breakdown of that compensation. The latter enormously increases the apparent frequency and importance of dilatation of the auricle in mitral stenosis cases, while the dilatation largely masks the signs of preceding hypertrophy. That dilatation is chiefly a breakdown phenomenon is indicated by the following consideration. The 70 cases to which I have referred included 18 which came from the surgical wards. In only one of these cases was dilatation of the auricle observed. In this case the stenosis was consider-

¹ Fagge and Pye-Smith, *Principles and Practice of Medicine*, Third Edition p. 74.

able (6 centimetres); hypertrophy was as marked as dilatation, and the patient died of his heart affection, with thrombosis and gangrene.

Now, it is reasonable to suppose that if this patient had died earlier in the course of his heart disease of some surgical malady, as most or all of the other surgical patients did, dilatation of the auricle would also not have been observed in this case. In 3 instances the left auricle was recorded as hypertrophied, only once, however, considerably; but as the stenosis was not severe, except in 2 of the 18 surgical cases, hypertrophy of the auricle was not, in my opinion, to be expected. In 29 of the 70 cases the stenosis was of the less severe form, the mitral measurement having exceeded 55 millimetres.

The condition of the auricle was reported on in only 12 cases. In only 1 case was the auricle markedly hypertrophied (a great contrast to the result observed in the severe stenosis cases given above), in 2 cases it was slightly hypertrophied, in 4 it was dilated and hypertrophied, and in 5 it was dilated without hypertrophy. Since in 17 of the 29 cases of slight stenosis the condition of the auricle was not recorded, it is probable there was no marked change to record.

The absence of auricular dilatation in the surgical ward cases, its absence likewise in the majority of medical ward cases of slight stenosis leaves very little basis for the common assumption that in the earlier stages of mitral stenosis the left auricle dilates.

The above statistics, which I have carefully collected and assorted clearly indicate, however, that as the stenosis increases and becomes severe the left auricle hypertrophies, generally (even though judged from *post-mortem* statistics) without dilatation, but sometimes accompanied or followed by dilatation. The latter is best explained as causing and accompanying auricular breakdown and as being an old-age phenomenon in the history of mitral stenosis.

NOTE ON MEDIASTINAL FRICTION.

By ARTHUR T. DAVIES, M.D., F.R.C.P.,

Physician to the Royal Hospital for Diseases of the Chest, London.

In a paper recently read before the British Medical Association, Dr. George Perez, of Teneriffe, describes a new auscultatory sign, which he discovered in a patient of his, who for nine years had been subject to attacks of mediastino-pericarditis. The two following cases, both of which were seen by Dr. Perez with me, illustrate very well this new sign.

R. N., aged 25, was admitted into the Royal Hospital for Diseases of the Chest, on July 1st, 1896, on account of dyspnoea and cough, due to mitral regurgitation, aortic stenosis and regurgitation with dilatation of the aorta, and signs of adherent pericardium. The patient had had three attacks of rheumatic fever. On raising the left arm to the vertical position above the level of the head, distinctly crackling or friction crepitations were audible along the left side of the sternum, and similar sounds were elicited on the right side of the sternum, when the right arm was raised.

The second case was that of B. C., aged 53, who was admitted under my colleague, Dr. Hensley (to whom I am indebted for allowing me to use the case) on June 10th, 1895, for pains in the chest and dyspnoea. He had suffered from intermittent fever, but denied syphilis. There was present a distinct aneurysm of the first part of the arch of the aorta. Raising either arm to the vertical position caused distinct friction crepitation sounds to be heard on the corresponding side of the sternum. In both patients lowering the arms also brought out these signs, but they were not audible during inspiration or synchronous with the heart's action. The explanation furnished by Dr. Harris, of Manchester, as to the cause of these sounds seems to me to be the most probable one. He attributes them to the stretching, by the raising of the arms, of adhesions situated round the vessels external to the pericardium.

PRESENTATION.—Dr. J. R. Kaye, medical officer of health to the West Riding County Council, has been presented with an illuminated and framed address, which sets forth the appreciation entertained by the Huddersfield Town Council of his work in that borough.

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, Etc.

HEAT APOPLEXY: HYPERPYREXIA: ICE BATH: RECOVERY.

WHILST going through the Red Sea, about 9 A.M. on the morning of October 21st, *en route* to Hong Kong, the ship's cook, a healthy looking man, was carried to the hospital with the report that he had suddenly fallen down when at work. He was quite sensible on admission, and answered questions, but his temperature was 104° F. He was immediately placed in bed, some diaphoretic mixture given, and an icebag applied to the head and nape of the neck. An hour after this I found him in much the same condition, the temperature being the same, and although he complained of headache he still answered questions sensibly. Ten minutes after, when discussing the case with Surgeon-Major F. M. Baker, the orderly reported the man's temperature as being 107.4° F. To make certain that this was correct I immediately after had the temperature again taken with the same result—107.4° F. The patient now got rapidly delirious, and he quickly fell into a comatose condition: the eyelids drooped, the pulse was quick and almost imperceptible, there was stertorous breathing, and there was also a cyanotic appearance of face and lips. The case now being diagnosed as one of heat apoplexy, and as it was evident to my colleague and myself that the man was rapidly sinking, a bath of iced water was immediately ordered. Whilst the bath was being prepared, solid lumps of ice were kept assiduously applied to almost every part of the body. Slight signs of reaction now set in, and, the bath being ready, he was at once placed in it, the iced water being freely sponged all over the body. The treatment employed proved most successful. Within eight minutes the temperature fell to 99.4°, and he was then taken out of the bath. Reaction set in rapidly, and he began to recognise people. The temperature again rose for a short time to 103°, but the application of ice to the head and neck, and wet sheets to the body, speedily reduced it to 100°, about which it kept for two days, and then fell to normal. In less than a week he was practically convalescent, without a bad symptom, the only other treatment being the administration of 10 gr. of calomel.

I think the above case worthy of record owing to the fact of its having occurred in the Red Sea, when the average temperature in the cabins was about 90° F., and the temperature of the galley in which the man worked had registered 122° F. Fortunately we had an unlimited supply of ice, but notwithstanding this, considerable difficulty was experienced in getting the temperature of the bath water reduced, owing to the very high temperature of the surrounding air.

Colombo.

R. C. GUNNING,
Surgeon-Major A.M.S.

ANEURYSM OF THE ASCENDING AORTA: RUPTURE INTO PERICARDIUM.

ON November 4th, 1896, I received an urgent message to visit a man who had been taken suddenly ill at a colliery. During the three months he had worked at the colliery he had done his full time of nine hours. After finishing his full day's work he had to walk a distance of half a mile underground; the last part of this distance was up a very steep incline. On the day of his death, when he reached the top of this incline, he remarked to another workman, "That is a steep hill." Having said this, he immediately fell down, and, after breathing heavily for two minutes, died. The man, whose age was 52, had frequently been in my surgery during the previous two weeks for medicine for his wife, and looked a remarkably healthy man.

His wife and two of his brothers informed me that they never knew him to have a day's illness in his life, and never to complain of being tired with his work; also that he was a very steady and abstemious man.

I made an inspection of the body on November 5th. On opening the chest I found the pericardium greatly distended. I carefully opened it, and measured the contents, which nearly filled a quart basin with clots and serous fluid. There was