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II.

TRAUMATIC RUPTURE OF THE VALVES OF THE HEART.

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The question whether external violence or a sudden strain of the body can cause a rupture of the valves of the heart is a very interesting one. Theoretically it seems possible that any sudden effort, which causes an intense and universal muscular contraction throughout the body, might result in such a great and sudden increase in arterial tension as to cause a rupture of the delicate semi-lunar valves, especially when at the same time the body, and in particular the aorta, already put on a stretch by the increased arterial tension, receives a violent jar. The result of daily experience, however, shows that such an accident is very uncommon, even in the case of

the aortic valves, which are much more exposed to such an injury than the mitral valves. Very few cases of such accidents have been reported, and the cases are extremely rare which show with any probability that healthy valves are ever the seat of traumatic rupture. The following case is one in which a sudden muscular effort seems to have caused a rupture of aortic valves, which had previously exhibited no symptoms of disease.

R. O. K., æt. 40. In November, 1883, the patient, who was tall and thin, but muscular, and had always been healthy and had never had rheumatism, lifted a heavy box from a shelf on a level with his head and placed it on the floor. As he did so he felt something give way in the front of his chest, and felt something fluttering and jumping inside of him. He felt so faint and weak that he had to be taken home in a carriage, and was confined to his bed for a couple of weeks. He never was able to resume work, and for the most part was confined to the house, rarely being able to take a short walk. In March, 1884, he fainted and

remained in a condition of collapse for several days, recovering from it very slowly. At that time his heart was examined. The area of cardiac dullness was slightly enlarged, and a loud, smooth diastolic murmur was heard over the aortic valves, and was propagated downwards towards the apex. No other murmur could be detected either over the heart or the vessels of the neck. The pulse was feeble and rapid. He had one or two more fainting fits, and on the evening of December 26, 1884 (a little more than a year after the accident), just after getting into bed, he suddenly died without any warning.

Unfortunately there was no autopsy held in this case, but it seems very probable that the suddenly increased arterial tension caused by the muscular effort of lifting the box down from the high shelf had torn one of the aortic valves; for immediately afterwards he had severe palpitation and symptoms of heart disease, which persisted up to the time of his death. In accordance with this supposition was the fact that the only murmur heard was that of aortic regurgita-

tion, and the lesion could not have been of long standing, as there was no cardiac hypertrophy. Of course, no absolute proof can be offered in this case that the aortic valves were perfectly healthy at the time of the muscular exertion, but certainly up to that time there were no symptoms of heart disease, and ever afterwards such symptoms were extreme. The mere fact that the symptoms of heart disease did not appear till after the accident is not in itself alone a sufficient proof that there was not previously existing heart disease, as is illustrated by the following case.

J. L., æt. 43. In October, 1887, the patient, who was a strong, muscular man, and had always been well, had never had rheumatism, and had never noticed any dyspnœa on exertion, was kicked by a horse. (The scar of the kick still remains over the middle of the left biceps muscle.) He was knocked about ten feet, striking on the right buttock. He was not confined to the house, but did not work for three weeks on account of his arm. At the end of three weeks he resumed work, and then for the first

time noticed that his breath was short, and he found that he became so weak that he was unable to work. He had no palpitation and no pain in the chest. An examination of the patient was made on November 17, 1887, at which time the area of cardiac dullness was much increased, commencing above on second rib, running one inch to the left of the left nipple and one inch to the right of the right border of the sternum. The heart's action was violent. A loud double blowing murmur heard over the whole front of the chest and faintly over the back, being loudest in the second right intercostal space close to the sternum. There was a strong systolic thrill in both subclavians, and a double murmur in the subclavian and also in the femoral on deep pressure. Some fine moist râles at base of chest on both sides. A sphygmographic tracing of the pulse is shown in the accompanying figure. The patient steadily failed in

strength, and suddenly died without any warning on January 10, 1888, about three months after the accident. At the autopsy, which was held thirty hours after death, the heart was found immensely hypertrophied and dilated. Although greatly dilated, the wall of the right ventricle was more than an inch thick. Except for a small calcareous plate at the base of one of the aortic valves, which did not interfere with its function, all the valves were normal, and the muscular tissue seemed healthy. The mitral orifice was dilated, admitting the ends of three fingers easily, and the aortic orifice was decidedly dilated. The arch of the aorta was also the seat of a great but uniform dilatation, and the intima was everywhere yellow and irregular, like the skin of a rough orange.

In this case the symptoms were due to a dilatation and loss of elasticity of the aorta and an enlargement of the aortic orifice, which were of such old standing that they had caused an extreme degree of cardiac hypertrophy, which in turn had made an apparently perfect compensation for the aortic lesion until after the

accident, and probably in consequence of this latter, this compensation was destroyed.

Although the symptoms of heart disease in this case did not appear until after the accident, yet there were many things which made it clear that this was not a case of traumatic rupture of the valves. In the first place, dyspnœa and weakness made their first appearance three weeks after the accident; in the next place, there was a double aortic murmur; whereas, if the aortic valves had been torn, there should have been a regurgitant murmur only; then, the sphygmographic tracing was not that of aortic regurgitation; and, finally, there was an extreme degree of cardiac hypertrophy, which showed that there was a heart lesion of much earlier date than the time of the accident.

This second case, then, teaches us that we must be cautious in considering cardiac symptoms which appear after a severe injury as necessarily due to the injury, and therefore of a traumatic nature, but it does not in the least invalidate the claim of the first case to be

regarded as a case of traumatic rupture of the aortic valves. The question as to the traumatic nature of the first case has a decidedly practical bearing, inasmuch as the patient had an accident insurance policy on his life. The claim, I believe, was never pushed by the family, and was never paid by the company, and yet strong proof could have been offered that his death was due to an accident.