

the cardiac lesions which involve a liability to sudden death.

Assuming correctness of the diagnosis, encouragement in the prognosis may be derived from cases in which the lesion existing in a considerable degree is remarkably tolerated. Some years since, a specimen showing rupture of the heart was exhibited at a meeting of a medical society, the rupture arising from fatty degeneration which was great and extensive. The rupture occurred during an attack which resembled *angina pectoris*. Up to this attack the patient had considered himself well, and took active exercise without inconvenience. He had no symptoms leading to the suspicion of any disease of the heart. It is not uncommon in autopsies to find more or less fatty degeneration of the heart when it had not been suspected, death having taken place from some intercurrent affection. These facts warrant hopefulness, as regards the prolongation of life, with a certain measure of health, for an indefinite period, even when symptoms and signs denote much fatty degeneration.

In treating of the prognosis in chronic diseases of the heart, some consideration of the liability to sudden death, should not be omitted; and my concluding remarks will relate to this topic.

In a very large majority of the cases in which the heart is the seat of organic disease, the cardiac lesions are not exclusively or directly, the cause of death. Most patients perish from superadded or intercurrent affections which may be either incidental to, and dependent upon, the disease of the heart, or accidentally associated with it. Of the cases in which cardiac lesions are fatal of themselves, that is, in consequence solely of their pathological effects, sudden death occurs in a very small proportion. As already stated, the popular impression is quite the reverse of this; and it is certain that many physicians participate, to a certain extent, in the common belief. The error is sustained by the frequency with which sudden death is attributed to disease of the heart on medical testimony, and after *post-mortem* examinations. It is too much the custom to refer the death to the heart whenever there is cardiac lesion, either from signs during life, or the appearances in the cadaver. But diseases of the heart, in a certain proportion of cases, do destroy life suddenly. What, then, are the lesions and the circumstances which render patients liable to sudden death?

The affection just noticed, namely, fatty degeneration, may be first mentioned. This affection involves a certain amount of liability to sudden death, rupture being the immediate cause in some cases, but oftener paralysis of the heart from over-

distention. Other things being equal, the liability is, of course, proportionate to the degree and extent of the degenerate change; and the amount of the disease can only be determined approximately by symptoms and signs denoting permanent weakness of the heart's action.

Of the valvular lesions, those which occasion free aortic regurgitation involve by far the greater liability to sudden death. The *rationale* is intelligible. The immediate cause of death is paralysis of the left ventricle from overdistention. The interesting fact that mitral regurgitant lesions are conservative, as regards the liability to sudden death from aortic regurgitation, has been stated in another connection. The fact of aortic regurgitation is determined by a diagnostic murmur; but the danger has relation, not to the existence of regurgitation, but to its amount. The latter is estimated by the increased size of the heart, the feebleness or extinction of the aortic second sound, and by the movements of the arteries which, together with certain characters of the pulse, denote that the regurgitation is considerable. It is rare for sudden death to be caused by aortic regurgitation so long as the heart is enlarged by predominant hypertrophy; generally, the weakness due to dilatation is a causative element.

A French writer, Mauriac, has offered an explanation of sudden death, in cases of aortic insufficiency, which is, perhaps, worthy of being considered. It is claimed, as preliminary to this explanation, that the blood is forced into the coronary arteries, not by the direct action of the left ventricle during the systole, but by the recoil action of the aorta directly after the ventricular contraction. The state of contraction of the muscular walls during the systole is supposed to constitute a mechanical obstacle sufficient to prevent, at this time, the entrance of the blood into the arteries of the heart. Now, assuming this, if there be much aortic insufficiency, a regurgitant current, caused by the recoil of the arterial coats, takes place, and, owing to the defect of that resistance which is afforded by the semilunar valves in health, the current into the coronary arteries is diminished. In this way, aortic regurgitation involves, in proportion as it is free and abundant, a diminished supply of the arterial blood to the walls of the heart; and, of course, the supply becomes more and more diminished in proportion as the systole of the ventricle is weakened by overdistention or other causes the arterial recoil being weakened in a corresponding degree. Mauriac would ascribe the sudden death to the want of arterial blood in the muscular walls, rather than to paralysis of the ventricle from