

overdistention. The latter is the *rationale* which I have given, and it is, I believe, correct; but it seems very probable that Mauriac has called attention to a condition which is important as contributing to the occurrence of sudden death. Mauriac's explanation has relation to a topic to be presently noticed, namely, occlusion of the coronary arteries as a cause of sudden death.

Dilatation of the right ventricle resulting from mitral obstructive, or regurgitant lesions, involves some, but a very small, liability to sudden death. The explanation is paralysis from overdistention. Probably the so-called "safety-valve function," at the tricuspid orifice, is a conservative provision against an accumulation of blood in the right ventricle sufficient to destroy life suddenly. It is hardly necessary to say that fatty degeneration of the heart, coexisting with valvular lesions and dilatation, increases the liability to sudden death; but it is not easy to determine this combination during life.

There is danger of sudden death whenever paroxysms of angina pectoris are associated with organic disease of the heart. Other things being equal, the danger is especially great when the angina is associated with aortic lesions which occasion free regurgitation, the mitral valves being sound; and angina is oftener associated with aortic than with mitral lesions. The association with fatty degeneration is also especially dangerous. These facts are easily understood when it is considered that aortic regurgitant lesions and fatty degeneration of the heart involve a liability to sudden death irrespective of angina. What causative agency is exerted by angina in addition to the lesions with which it may be associated? This question can perhaps now be answered satisfactorily, with our knowledge of the effect upon the movements of the heart of galvanism transmitted through the pneumogastric nerves.

Facts appear to show that the force and regularity of the cardiac movements depend on an innervation received through the pneumogastrics. The division of these nerves is followed by notable perturbation of the action of the heart, its movements becoming rapid and feeble. A feeble galvanic current suffices to arrest its action, producing in effect paralysis. Without entering into any discussion of the explanation of these facts, they lead to the rational supposition that, in certain cases of angina, there is superadded to the neuralgic pain a morbid innervation exerted through the pneumogastrics, producing the perturbation of the heart's action which is of frequent occurrence, and sometimes

arresting the movements of the heart like the galvanic current.

I have heretofore held the opinion that paroxysms of angina pectoris involved a liability to sudden death, only where there are lesions, more or less serious, of the heart or aorta; and hence, wherever from the absence of physical signs organic diseases could be excluded, we are warranted in giving positive assurance of the absence of danger. As a rule, I believe still that this opinion is well founded; but within a short time I have learned by experience that there may be exceptions to the rule, and that the opinion is therefore, in individual cases, to be expressed with a certain amount of reserve. Not long since I saw, with a member of this society, Robert F. Weir, a patient who suffered from angina pectoris. A physical examination revealed no signs of disease of the heart or aorta. The patient, however, died suddenly in a paroxysm. On an examination, *post mortem*, there were found some dilatation of the aorta, and some calcareous deposit; but the valves were sufficient, and the heart was neither enlarged or fatty. It was evident that the lesions had nothing to do with the sudden death, except, perhaps, as entering into the causation of the angina. This case would seem to show that angina may destroy life suddenly, by an inhibitory or paralyzing effect upon the heart, irrespective of cardiac lesions.

The inquiry arises, What are the symptoms during a paroxysm of angina which denote danger of sudden death? In answer, it may be said that there is absence of danger so long as the action of the heart is but little or not at all disturbed, whatever lesions exist, or whatever lesions are absent or present. There is little or no danger if the patient have not a sense of impending death, and if the necessity of perfect quietude be not felt. On the other hand, the danger is great in proportion as the action of the heart is rapid, feeble, irregular, or notably retarded.

Is sudden death ever attributable to either embolism or thrombosis of the coronary arteries? It was considered that this might have been the explanation of the sudden death of the late Prof. Enos, of Brooklyn. In that case both coronary arteries were obstructed by calcareous masses, and it was conjectured that, the obstruction taking place suddenly, an arrest of the circulation in these vessels caused paralysis of the heart. It may be assumed that defective nutrition and consequent weakness resulting from obstruction of the coronary arteries, whether due to an embolus, or a thrombus, or the encroachment of calcareous deposit upon the mouths of the vessels, contributes to sudden death,