Medical Matters.

ANGINA PECTORIS.

Sir William Osler, M.D., F.R.S., Regius Professor of Medicine in the University of Oxford, in the second of the Lumleian Lectures on Angina Pectoris, delivered before the Royal College of Physicians, of London, and published in the *Lancet*, spoke in part as follows: At the outset let us frankly face certain obscurities which have not yet been cleared up. Why is it more common in the upper classes? Why do we not see it more often in hospital practice? Worry and work are the lot and portion of the poor, among whom vascular degeneration is more widespread. It is as though only a special strain of tissue reacted *anginally*, so to speak, a type evolved amid special surroundings or which existed in certain families. Or there may be a perverted internal secretion which favours spasm of the arteries, as Harvey at Cambridge has shown to be the case with pituitary extract and the coronary vessels. And a case of aortic valve disease is reported in which the use of this extract caused anginal attacks. This suggestion is supported by the fact that in myxœdema anginal attacks may be caused by thyroid extract. It is not the delicate neurotic person who is prone to angina, but the robust, the vigorous in mind and body, the keen and ambitious man, the indicator of whose engines is always at "full speed ahead." There is, indeed, a frame and facies at once suggestive of angina—the well "set" man of from 45 to 55 years of age, with military bearing, iron-grey hair, and florid complexion. . . Still more extraordinary and inexplicable is an imitative feature, if one may so speak of it, by which the repeated witnessing of attacks may induce one in the observer. There are two primary features of the disease, pain and sudden death—pain, paroxysmal, intense, peculiar, usually pectoral, and with the well-known lines of radiation—death in a higher percentage than any known disorder, and usually sudden. Often, indeed, it is, as the poet says, "Life struck sharp on death." The problems for solution are: What is the cause of the pain? Why the sudden death? The secondary features of the attack, the vaso-motor phenomena, the radiation of the pain, the cardiac, respiratory, and gastric symptoms are of subsidiary interest.

After discussing exhaustively the morbid anatomy, the involuntary muscle pain, the cardio-vascular pain, and the arterial spasm, Professor Osler said: After all this talk, what in a few words is a reasonable explanation of the pain in angina? Angina results from an alteration in the working of the muscle fibres in any part of the cardio-vascular system, whereby painful afferent stimuli are excited. Cold, emotion, toxic agents interfering with the orderly action of the peripheral mechanism, increase the tension in the pump walls or in the larger central mains, causing strain, and a type of abnormal contraction enough to excite in the involuntary muscles painful afferent stimuli. Mackenzie suggests that there is rapid exhaustion of the function of contractability, which is after all only the *fatigue* on which Allan Burns laid stress; but I feel that in disturbance of this Gaskellian function is to be sought the origin of the pain, whether in heart or arteries. . . *In stretching, in disturbance of the wall tension* at any point, and in a pain-producing resistance to this by the muscle elements, lie the essence of the phenomena.

What is the explanation of the sudden death? There are three modes of dying in angina pectoris. The one which specially interests us here is the form which, as Walshe says, "is sudden, instantaneous, coeval, with a single pang." No form of death so placid, so peaceful, and so much to be envied, as it probably is without a pang. The functions of life appear to stop abruptly, with a gasp or two all is over. It is extraordinary how little a man may be disturbed in this death. An old doctor whom I knew well stopped at his house to write a prescription. With pen in hand he died at the desk, where I found him, as if in sleep, with his head peacefully on his arm and pen in hand. Another friend, the subject of angina, whom I had only left a few minutes previously, talking quietly to Dr. Thayer, fell over on his bed; both pulse and breathing seemed to stop simultaneously. It must be a vagal death, a sudden inhibition of the inspiratory centre in the medulla. It is exactly paralleled in chloroform death, when the inspiration stops abruptly, while the heart may continue to beat. In a third case the patient gave a sudden cry, clasped his hands over his heart, the eyes became fixed, and he fell over dead after giving two inspiratory gasps. No pulse could be felt at the wrists, but feeble heart sounds could be heard for three minutes.

A second mode of death is also seen in which, following a series of severe attacks, the heart grows gradually feebler, and the patient dies in progressive asthenia, often with Cheyne-Stokes respiration. And thirdly, a certain number of patients die in the cardiac complications, and it is interesting to note how after great misery, caused by repeated attacks, when cardiac insufficiency is established, even with the dyspnœa, the patient is much happier, and dies slowly, if not so suddenly and placidly.

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