SURGICAL TREATMENT OF ANGINA PECTORIS

INGA LINDGREN, M.D., AND HERBERT OLIVECRONA, M.D.

Neurosurgical Clinic, Serafimerlasarettet, (Director: Professor H. Olivecrona) and IVth Medical Service, St. Erik's Hospital (Director: Professor H. Berglund), Stockholm, Sweden

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The extensive researches of Blumgart have shown that the structural basis for coronary pain is an arteriosclerotic process with obstruction or obliteration of one or more coronary arteries before an adequate collateral circulation has been developed. On the other hand, if the development of the collateral circulation has kept pace with the progressive arterial narrowing, myocardial damage and pain are absent even if one or more of the coronary arteries are obstructed.

If the increased blood supply to the myocardium demanded by muscular exercise cannot be delivered through the ordinary or collateral channels, coronary pain results. A short rest is usually sufficient to restore the circulatory balance and the pain subsides. Dilatation of the coronary arteries by nitroglycerine hastens the process and may, if the drug is taken before muscular exertion, prevent an anticipated attack.

Vasoconstriction, on the other hand, is an important factor in anginal pain. Practically every sufferer from angina pectoris is worse in the winter; his capacity for muscular work is much less in cold weather. Sudden cooling from any cause is frequently sufficient to bring on an attack. Freedberg, in a series of experiments, has been able to show the effect of cooling upon the exercise tolerance in angina pectoris. He found by testing the same patient's capacity for exercise at different temperatures that the tolerance was much less at temperatures of 7–13°C. (45–55°F.) than at 24°C. (75°F.). His investigations thus lend experimental support to clinical experience that cold weather has a bad effect on sufferers from angina pectoris. Since the increased cardiac work due to rise in blood pressure and tachycardia is relatively slight under the experimental conditions used by Freedberg, it is hardly possible to escape the conclusion that the effect of cold is due to reduction of the coronary blood flow because of vasoconstrictor impulses to the coronary arteries.

The importance of vasoconstriction is further emphasized by the effect of emotion upon patients with angina pectoris. Practically every patient will say that anger or any other emotional upset is likely to provoke an attack. Mainzer has shown that the fear before operation may cause transient electrocardiographic changes indicating coronary vasoconstriction, not only in patients suffering from coronary disease but also in persons with normal hearts, especially in neurasthenics. There are many cases on record of patients dying suddenly from coronary occlusion after a heated argument,
the most famous case being that of John Hunter. Anybody who has seen a number of patients with angina pectoris can probably recall such instances. It also seems possible that the pain provoked by effort or emotion might induce further vasoconstriction establishing thereby a vicious circle. Experimental work has lent support to this opinion. Gold and Gilbert, amongst other investigators, have shown that spontaneous or experimentally induced extracardiac pain produces electrocardiographic changes in patients suffering from angina pectoris.

Further it has been shown that cardiac pain can be ameliorated or abolished by local anaesthesia of the area of referred pain. During a functional load placed upon the heart pain is diminished or completely abolished after precordial anaesthesia, while the same functional load produces typical anginal pain in the same individual unanaesthetized. Electrocardiograms registered during the test with anaesthesia show less pronounced changes than during a corresponding test without anaesthesia. This may indicate that amelioration of anginal pain is accompanied by improved coronary circulation.

The purpose of the neurosurgical interventions employed in the treatment of angina pectoris is to interrupt the sensory pathways of the heart or/and to block the pathways for vasoconstrictor impulses to the coronary arteries. There is considerable difference of opinion concerning the relative importance of afferent and efferent fibres in the vasomotor reflexes of the heart. We propose to discuss this point in a later section of this paper.

The afferent pathways between the heart and the brain are reasonably well known. These pathways run through the middle and inferior cardiac nerves, which join the sympathetic trunk and ganglia near the middle and inferior cervical ganglia. In the sympathetic trunk the afferent nerves from the heart take a downward course and leave the paravertebral chain of sympathetic ganglia through the white communicating rami to the first 2 or 3 thoracic nerves to enter the corresponding posterior roots, and after crossing over to the opposite side of the spinal cord reach the brain through the spinothalamic tract. A second important and constant pathway for afferent impulses is through the thoracic cardiac nerves, first described by Braeucker in 1927, and by Ionesco and Enachesco in the same year. The thoracic cardiac nerves run from the heart to the 3rd and 4th, possibly also to the 5th, thoracic sympathetic ganglion to enter the posterior root through the corresponding white rami communicantes. In addition there are probably accessory afferent pathways with wide variations in their individual development, as shown by the localization of referred pain to segments above or below the 5 upper thoracic segments. The phenomenon of "migration of pain" also indicates the presence of accessory afferent nerves. The anatomy of the accessory sensory connections is very imperfectly known. Referred pain below the 5th thoracic segment is on the whole very rare and migration of pain in our experience never occurs below this segment but always to cervical and cranial segments. This suggests that sensory impulses are