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A NEW CONCEPTION OF ANGINA PECTORIS

BY

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Before considering angina pectoris it is necessary to recall certain physiological facts about the course of the vasodilator and protopathic pain fibres. The vasodilator fibres, to both the somatic and the visceral organs do not run in the parasympathetic system, nor do they relay in the various sympathetic ganglia in the body. Apart from a few fibres in the parasympathetic system the only known vasodilator fibres are those of unmyelinated type running in the posterior and trigeminal nerve roots and having their cell bodies in the corresponding ganglia. Thus stimulation of the peripheral ends of the posterior nerve roots produces vasodilatation in the corresponding dermatome and also in the deep tissues and viscera, and herpes zoster in the skin. Moreover, providing the sino-aortic nerves are intact, the raising of the blood pressure in the carotid artery or aorta in a sympathectomized animal still produces a normal reflex vasodilatation in other parts of the body (Bayliss, 1901, 1902). Lewis (1927) further showed that if the posterior roots are cut distal to their ganglia and their peripheral ends allowed to degenerate, the arterial "flare" of the "triple response" in the skin fails to occur after the application of noxious stimuli. This reactive hyperaemia occurs in all tissues when injured or stimulated (Love's reflex).

The whole of the sympathetic system may be removed and yet reflex vasodilatation will still occur in response to a stimulus to a tissue. A stimulus or injury to any tissue, including anoxia, must therefore result in a reflex activity in the unmyelinated posterior root vasodilator nerve fibres. These are, of course, the same fibres which conduct protopathic pain impulses, and are cholinergic in type. Such pain fibres supply the visceral and somatic blood vessels and run in the posterior nerve roots (Moore and Singleton, 1933). They are evidently the vasodilator fibres of these vessels. It has now been shown that an excessive arterial vasodilatation results in the experience of protopathic pain. This is seen, for example, during a migrainous attack and in cases of erythromelalgia. In the former, hanging the head down, which normally causes arterial vasodilatation, will also greatly exaggerate the pain. In the latter condition the affected limb exhibits arterial vasodilatation and is painful. Dependence of the limb increases both the vasodilatation and the pain, while elevation has the opposite effect. Thus the passage of excessive numbers of impulses in vasodilator fibres results in painful experiences.

Very many theories regarding the cause of anginal pain have been put forward. Allbutt (1915) championed the tam-bour theory, which supposed the pain to be due to bulging of the suprasigmoid portion of the mediastinum. This theory has been discarded, and it is now fashionable to ascribe all anginal pain to ischaemia of the cardiac muscle, due either to coronary arterial disease, anoxia, or anaemia, or to a low pulse pressure and so diminished flow in the coronary vessels. Anoxia is thought to cause the accumulation of metabolites, such as the P factor of Lewis (1931), which stimulate the nerve endings lying on the coronary vessels. Yet in some cases there is no evidence of coronary disease, anoxia, anaemia, or low pulse pressure, and it is then usual to invoke the hypothetical phenomenon of spasm in the coronary vessels to account for the anoxia thought to produce pain. There are many reasons to doubt that ischaemia or anoxia is always the cause of angina from cardiovascular disease. For example, severe coronary

Air transport and packing in dry ice were recently used for the first time to convey dried cultures of *Staph. pyogenes* in glass ampoules and bacteriophage in fluid from the Central Public Health Laboratories in London to Sydney, Australia, whence they were delivered to Miss Nancy Atkinson, bacteriologist at the Institute of Medical and Veterinary Science, Adelaide. B.O.A.C. staff renewed the ice at Lydda, Calcutta, Singapore, and Sydney. The journey took about four days.

disease and even complete occlusion of a large coronary vessel may be quite painless. Some other factor must exist to account for the occurrence of pain in some cases and not in others.

Anginal Pain and Normal Heart

There have gradually accumulated many accounts of patients with typical anginal pain from disease of tissues in the neighbourhood of the heart and in whom the heart is normal. These include:

Acute Mediastinitis or Mediastinal Abscess.—This results in severe pain substernally and between the shoulder-blades, and it may also pass down the arms and into the neck. Allbutt described a case of tuberculous mediastino-pericarditis with such symptoms.

Mediastinal Tumours.—These may cause pain substernally, between the shoulder-blades, and passing into the arms. It may be constant, intermittent, or paroxysmal (French, 1924).

Pneumo-mediastinum.—The presence of air in the mediastinum may cause temporary and often paroxysmal excruciating substernal pain passing into the arms, neck, jaws, and teeth (Scott, 1937).

Para-oesophageal Hernia of Stomach into the Mediastinum.—Roesler (1939) states that this may cause typical angina. An example of this is the following case.

Case 1.—A woman aged 57 attended the Royal Cancer Hospital under the care of Dr. P. E. T. Hancock on Nov. 18, 1947. Two years previously within a few days she had developed epigastric and substernal pain passing to the interscapular region and into both arms, especially the left. The pain came on about a quarter of an hour after meals, lasted about an hour, and was sometimes accompanied by vomiting. In addition she had periodic attacks of numbness and tingling in one or other digit of the left hand quite apart from the anginal attacks. The pain disappeared after two months, but had returned a month before attendance. Its features were the same as during the previous attack. It was of considerable severity and made her afraid to eat. Three weeks previously she had vomited a small amount of "coffee-ground" material. Her appetite had disappeared and she had lost 7-8 lb. (3.17-3.62 kg.) in weight. On examination, apart from some slight tenderness on palpation in the middle of the epigastrium, no abnormal physical signs were to be made out. The blood pressure was 140/80 mm. Hg. No abnormal degree of peripheral arteriosclerosis was present and the cardiac apex beat was not displaced. The electrocardiogram was normal. A barium meal, however, showed the presence of a para-oesophageal hernia of the fundus of the stomach into the mediastinum to the left of the cardia. A fractional test meal revealed hypochlorhydria. The blood count showed: red cells, 4,040,000 per c.mm.; Hb, 62%; C.I., 0.77. She was treated under an ordinary peptic ulcer regime, including 10 minims (0.6 ml.) of tincture of belladonna half an hour before meals. Within two months her symptoms had completely disappeared and she had regained her former weight. A second barium meal showed no change in the radiological appearances.

Partial or Complete Oesophageal Rupture Within the Mediastinum.—Jackson and Jackson (1935-6) report that this causes typical angina. Mere distension of the oesophagus may have a similar effect.

A Dissecting Aneurysm of the Aorta in the Mediastinum.—While the dissection is taking place typical anginal pain may occur (Allbutt, 1915).

Spontaneous or Artificial Pneumothorax.—Anginal pain resulting from spontaneous pneumothorax has been described by Montaneda *et al.* (1937) and from artificial pneumothorax by Lord (1925), Ballon and Francis (1929), Hamman (1934), and others. Obviously a unilateral pneumothorax may cause considerable displacement of the mediastinum when the latter is slack.

Pneumonia.—Anginal pain may occur during an attack of pneumonia (Kelman, 1919, Wassermann, 1920). In this illness the mediastinum may either be displaced or be the seat of glandular enlargement and inflammation.

Aneurysmal Dilatation of the Pulmonary Artery.—Anginal pain resulting from such a lesion has been described by Henschen (1906), Bedford (1929), and others. The disease may occur in comparatively young people, when it is often congenital in nature and unaccompanied by anoxia. The coronary vessels may then be completely normal and there is no question of involvement of their ostia by disease. The pulmonary artery lies within the mediastinum, and during its enlargement presses on the mediastinal tissues.

Aneurysmal Dilatation of the Aorta.—Angina is very commonly associated with such a lesion even in the absence of any evidence of aortic insufficiency. It is usually attributed to co-existing coronary disease or to involvement of the ostia of the coronary arteries by the aneurysm or a syphilitic process. The electrocardiogram may show no evidence of myocardial disease. The mediastinal structures are always disturbed, however. Mechanical distension of the aorta in dogs failed to give rise to anginal pain (Sutton and Lueth, 1930), though this developed when the periaortic tissues were damaged. It seems probable, therefore, that disease of the artery wall itself is not responsible for the anginal pain, but that pressure on periaortic structures is important.

Diaphragmatic Flutter.—This may give rise to paroxysmal anginal pain passing to the arms (Winkler, 1933; Hofbauer, 1933; Porter, 1936). The electrocardiogram may be normal between the attacks but show an inverted T wave during them.

Pressure on the Diaphragm by Distension of the Stomach or Colon with Gas.—This may cause anginal pain localized to the left chest and arm (Lurjé and Stern, 1931; and others). It may perhaps displace the mediastinum.

Disease of the Gall-bladder.—This not uncommonly causes typical anginal pain, which may disappear when the gall-bladder is removed.

There is interference with the aeration of the blood in the lungs in many of the above conditions, as in cases of pneumonia or pneumothorax. A tuberculous mediastinal abscess is usually secondary to lung disease. A mediastinal tumour or an aneurysm may likewise cause collapse of a portion of the lung. Yet the complete lung on one side may be removed without causing angina. Moreover, in such conditions as pneumomediastinum, para-oesophageal hernia of the stomach, aneurysmal dilatation of the pulmonary artery, disease of the gall-bladder, or mediastinitis there is no suggestion of anoxia or of disturbance of the coronary arteries. In all the above conditions there is either disturbance of the mediastinal connective tissue by inflammation, pressure of one of the contained tubes or a gastric hernia, or displacement due to unilateral pressure of air in the pleural cavity, or there is disease of the diaphragm and the gall-bladder. Embryologically the connective tissue of the mediastinum, diaphragm, and gall-bladder all originate from the septum transversum. All such mechanical or inflammatory lesions cause a reflex arterial hyperaemia in the affected tissues. This means increased numbers of impulses in the appropriate vasodilator pain fibres in the posterior roots.

Other Causes of Anginal Pain

Apart from the above causes anginal pain may result from coronary sclerosis. It may also accompany hypertension (whether essential or renal in type or due to adrenal medullary or cortical tumours), aortic regurgitation or stenosis, mitral stenosis, and paroxysmal auricular fibrillation or tachycardia (see King, 1941; White, 1944). In such conditions the pain is usually brought on by physical exertion, flatulence, a heavy meal, or emotional upset. It may tend to occur during sleep. It is sometimes accompanied by flushing, and is occasionally followed by the appearance of herpes zoster in the segmental areas affected by the pain (see White, 1944). Anginal pain may also result from coronary thrombosis, acute cor pulmonale due to pulmonary infarction, cardiac trauma, the tearing of a papillary

muscle (Todd, 1848; Glendy and White, 1936; Payne and Hardy, 1937), acute toxic myocarditis, hydatid infestation of the heart (Evans, 1832), metastatic tumours of the myocardium (King, 1941), abscess of the heart, and many other inflammatory states of the myocardium. Such pain may also result from general disturbances, such as anoxia, anaemia, exposure to carbon monoxide, trichlorethylene ("trilene"), pentnucleotide, and nicotine, hyperinsulinism, Graves's disease, the injection of adrenaline, or anxiety states. It may constitute a tabetic crisis or occur as a withdrawal symptom in morphine or cocaine addicts.

The coronary vessels are surrounded by unmyelinated fibres, which accompany them to the various tissues of the heart. Some of these fibres must conduct vasodilator impulses. It is well known that the exposed heart is insensitive to painful stimuli. Sutton and Lueth (1930) showed that cutting, piercing, tearing, or pinching the myocardium or distension of the ventricle did not give rise to pain. On the other hand, tightening a ligature around the coronary vessels resulted in pain referred to a limb. Such a ligature included the unmyelinated fibres lying on the coronary vessels. Painting the vessel proximal to the ligature with 80% alcohol before this procedure prevented the onset of pain.

Gorham (1943) showed that tension on the coronary vessels may cause cardiac pain without producing any electrocardiographic signs of ischaemia. This suggests that the impulses giving rise to pain originate in the walls of the coronary arteries. By analogy with other blood vessels it would be expected that cardiac pain would result from excessive activity in coronary vasodilator fibres running in the posterior nerve roots. This is confirmed by the following observations.

1. Anginal pain is abolished by section or blocking of vasodilator pain fibres of D 1-4 (5) on the corresponding side.

2. A lesion of any tissue of the body, including the heart, leads to local arterial vasodilatation, whether the lesion is due to infarction, trauma, or an infective agent. Exposure of an organ to anoxia, anaemia, or carbon monoxide has the same effect. This vasodilatation results from increased activity in the vasodilator pain fibres. When the agents affect somatic tissues pain results. When they cause dilatation of the pial and cerebral vessels a headache may appear. When they affect the heart they may cause angina. Thus infarction often leads to anginal pain and changes in the electrocardiogram. Trauma to the heart may likewise cause anginal pain and changes in the electrocardiogram similar to those of coronary thrombosis (King, 1941). Anoxia, anaemia, or carbon monoxide poisoning may have the same effects. Rupture of a papillary muscle which is followed by inflammation and vasodilatation leads to severe angina. Acute toxic myocarditis is accompanied by pronounced coronary vasodilatation and often substernal anginal pain. Bacterial and other infections and hydatid infestation involving the heart may also cause angina (King, 1941). In all these anginal states there occurs a transient or permanent coronary arterial vasodilatation. After infarction this is maximal in the first few hours, and it is at this time that the anginal pain is at its height.

3. A rise in blood pressure increases the work of the heart and so results in vasodilatation in the coronary vessels. It also produces a reflex vasodilatation throughout the body by way of the sino-aortic nerves. This is shown by the high colour of the skin of many hypertensives. The vasodilatation in the pial and cerebral vessels causes headache. This generalized vasodilator activity must augment the increased activity in the coronary vasodilator fibres caused by increased work. In such conditions of hypertension angina is common.

4. In tabes there is destruction of the pain fibres in the posterior nerve roots. A tabetic crisis is painful and is accompanied by vasodilatation in the affected organ or tissue. It may be followed by small haemorrhages. In some cases such a crisis may take the form of an anginal attack.

5. During the withdrawal of drugs from morphine and cocaine addicts there is a general vasodilatation in all organs, and angina pectoris frequently occurs.

6. An anginal attack often occurs in sleep when there is a general vasodilatation in the skin and other organs.

7. A heavy meal causes vasodilatation in the stomach and neighbouring tissues. It may induce an anginal attack in a subject of the disease.

8. An anginal attack may be accompanied by flushing of the face and upper part of the body—evidence of increased activity in vasodilator fibres.

9. Emotion often causes vasodilatation in tissues, and may induce an anginal attack in a subject of the disease or in one with a normal heart.

10. The injection of insulin not infrequently causes flushing of the skin and sweating (both signs of activity in cholinergic fibres), and one of its early effects is to produce increased gastric peristalsis, which is abolished by the application of atropine (Best and Taylor, 1943), and is thus due to activity in cholinergic nerve fibres. At the same time it often produces a migrainous or other type of headache. The hormone evidently stimulates cholinergic, including vasodilator, fibres. Not uncommonly it may induce an anginal attack, even in a person not normally subject to them.

11. In cases of Graves's disease there is increased activity of the heart, but also a notable vasodilatation in the tissues of the upper half of the body involving the mediastinum as well as the skin. Angina may occur on effort.

12. Nicotine likewise produces a general vasodilatation and not uncommonly headache. It is a well-known cause of anginal attacks.

13. Trichlorethylene, like other anaesthetics, causes general vasodilatation. It may induce angina, suggesting that the latter is due to excessive activity in vasodilator pain fibres.

14. In a case observed by me the intravenous injection of pentose nucleotide, a powerful vasodilator, into a patient with no sign of cardiac disease produced a typical attack of angina.

15. A coronary thrombosis with anginal pain may be followed by herpes zoster in the areas where the pain is felt. This indicates increased activity in vasodilator root fibres.

16. An attack of brachial radiculitis in a subject with no evidence of heart disease may cause angina of effort, which disappears when the radiculitis clears up. Such cases have been described by Nachlas (1934), Josey and Murphey (1946), and others. An example which I saw in the recent epidemic of brachial neuritis is here described.

Case 2.—An Army officer aged 26 was seen on Feb. 26, 1943. Three months before, while sleeping under canvas and exposed to damp, he developed severe aching and stiffness in the neck. His regimental medical officer diagnosed rheumatic torticollis. After heat and massage treatment in the next month the aching spread to the shoulders and down both arms, especially the left. The pain was particularly severe at night and kept him awake. On rising there occurred stiffness, redness, and swelling of the hands and neck, and crepitations in the shoulder-joints. The finger-tips tingled and felt numb. The arms felt weak and ached, especially after use. The symptoms persisted in spite of the heat and massage treatment. Three weeks before being seen he began to experience great distress on walking only 80 yards (73 metres) at an ordinary pace. This was accompanied by a constricted feeling round the upper thorax and a feeling of pressure under the upper sternum with pain, which passed down the inner side of both arms to the fourth and fifth fingers, especially on the left side. The pain was accompanied by a burning, tingling sensation in these fingers. The symptoms caused the patient to stop walking and would then pass off within about half a minute, though the burning and tingling in the fingers persisted considerably longer.

Examination showed a marked tenderness on pressure over the cervical and brachial nerve roots, which felt thickened. Pressure produced a tingling sensation in the fingers, which were somewhat swollen, with thickening of the interphalangeal joints. The hands and forearms were reddened and hot.

Power in the upper limbs was normal and there were no changes in the tendon reflexes. There was a marked hyperalgesia in the skin of the upper limbs and a distaste for handling of the limbs. No sensory loss could be made out. Crepitations were present on movement of both shoulder-joints. The drawing of a pin along the skin of the forearms and back of the hands produced an intensely disagreeable sensation and a gross exaggeration of the "flare" of the "triple response," which persisted for over an hour. In other parts of the body this procedure produced only a normal flare. A radiograph of the heart and cervical spine showed no abnormality, and an electrocardiogram was normal. The blood pressure was 120/80 mm. Hg and the pulse regular.

The patient was advised to rest in bed lying flat with the arms in slings, and acetyl-salicylic acid, 10 gr. (0.65 g.), was given three times a day. After a month the pains and the anginal symptoms had disappeared, though the hands were still swollen on waking in the morning. After a further month, using the limbs as little as possible, there was still slight swelling of the hands on waking. When seen a year later his symptoms had completely disappeared.

In this case there was no evidence of coronary disease, but the arm was painful and hot, and its tissues showed marked vasodilatation. The flare of the triple response was very easily obtained on the affected skin. This vasodilator nervous activity must involve deep tissues supplied by the affected roots and D 1-2 roots, supplying the coronary vessels.

Conclusion

In all cardiac conditions in which angina or coronary pain is experienced there is evidence of excessive activity in the vasodilator pain fibres passing to the coronary vessels, and this is evidently the origin of the anginal pain. Lesions of the derivatives of the septum transversum have also been shown to cause angina. Their nerve supply from the posterior nerve roots is established very early in development, and the nerve supply of the derivatives is therefore closely related if not identical. The mediastinal tissues are supplied with vasodilator fibres by way of D 1-5 posterior nerve roots, which also supply the heart. Lesions of these, as of other tissues, result in an increased activity in the vasodilator fibres passing in the same nerve roots as supply the coronary vessels. Hence any lesion of either mediastinum or heart which causes an increased activity in vasodilator pain fibres may result in angina. Such a conception brings angina into line with other painful conditions, such as headache, resulting from excessive vasodilatation in blood vessels. Thus angina is not directly due to anoxia or to vasoconstriction but to excessive attempts at vasodilatation.

The activity of any organ, including the heart, is accompanied by a reflex hyperaemia and by arterial vasodilatation, which occurs through the agency of vasodilator nervous impulses. The afferent nerve endings are stimulated by metabolites. Coronary sclerosis prevents vasodilatation during increased cardiac activity and allows the collection of metabolites. These cause excessive numbers of reflex impulses in the coronary vasodilator pain fibres in an attempt to produce further vasodilatation, and it is this excessive nervous activity which appears to give rise to the pain in such conditions.

Summary

Evidence is adduced to show that anginal pain may be caused by disease of both the tissues of the mediastinum and the heart. It seems to be due to excessive activity in the vasodilator pain fibres in the posterior nerve roots passing to the mediastinal structures and the coronary vessels, and it is not necessarily due to anoxia.

I would like to thank Dr. P. E. Thompson Hancock for permission to publish the details of Case 1.

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TOLERANCE LIMITS TO RADIANT HEAT

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Radiant heat, a combination of visible and infra-red radiations, plays an important part in the practice of physiotherapy. In spite of this widespread use there have been few studies on the optimal and curative effects of measured amounts of radiant heat. The usual method of regulating the dosage of radiant heat is briefly to adjust the source used (incandescent filament bulb or resistance-wire heating element) at a distance from the patient's skin so that he feels comfortably warm but will be free from any hazard of burn. Since the conditions under which a burn will occur are poorly defined, it is necessary always to err on the side of safety.

It is perhaps not generally realized that heat is more easily administered by radiation than by convection and that accurate dosage can be prescribed (Evans and Mendelsohn, 1945a). A series of investigations carried out in this department and at the Clarendon Laboratory, Oxford, have led to the development of a suitable instrument for measuring radiation flux, including all visible and infra-red radiation (Evans and Mendelsohn, 1944, 1945b). The aim of the present investigation was to use this thermoradiometer for the determination of tolerance limits of the