# SIR JAMES MACKENZIE'S HEART

BY

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WITH AN ACCOUNT OF HIS CLINICAL HISTORY BY JAMES ORR, AND NOTES ON THE PATHOLOGICAL HISTOLOGY BY D. F. CAPPELL.

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The following description of Sir James Mackenzie's heart has been prepared in accordance with his desire, expressed to myself and to other friends, that after his death his heart should be examined to ascertain what information it furnished upon the symptoms that he had experienced. He died in London on January 25, 1925, aged 72. Some weeks before his death he told Dr. John Parkinson that he wished him to make a post-mortem examination. This request was confirmed, after his death, by his brother, Sir William Mackenzie, now Lord Amulree. The examination was performed, some fourteen hours after his death, by Dr. John Parkinson, assisted by Dr. J. W. Linnell. The heart was removed and subsequently sent to me at St. Andrews for further examination. Dr. Parkinson noted that nothing abnormal was found in the pericardium.

In order to correlate the clinical symptoms with the pathological condition, it has been necessary to compile an account of Sir James Mackenzie's illness. This has not been easy for, like so many other doctors, he had not been under the care and observation of a medical man from the commencement of his illness. His own case is referred to both in his book on angina pectoris (Case No. 28) and in that on diseases of the heart, as well as in the Reports of the St. Andrews Institute.

While he was in London, from 1908 to 1918, he mentioned to Sir Thomas Lewis that his anginal history began suddenly. While he was in St. Andrews, from 1918 to 1924, engaged in founding the Institute for Clinical Investigation which bears his name, he was on many occasions examined by Dr. James Orr, and discussed with him his condition. Dr. Orr also saw him during several of the attacks of angina, from which he suffered with increasing severity as the years went on. After his return to London in 1924 he was not under the care of any medical man until very shortly before his death, when he was seen by Dr. Parkinson and Dr. C. M. Anderson.

Dr. Orr, who had seen him during the whole of his stay in St. Andrews, has written the account of the clinical history which follows. My colleague, Professor D. F. Cappell, undertook the histological examination of the blood vessels and heart muscle, and his notes on them are included in the description.

## CLINICAL HISTORY AT ST. ANDREWS

The medical life history of Sir James Mackenzie is the story of the onset and gradual progress of angina pectoris from sclerosis of the coronary arteries. He had a mild attack of typhoid fever in 1880 and an occasional attack of renal colic in his later years, but suffered from no other illness.

With the exception of a tendency to extrasystoles commencing at the age of forty, the first evidence of real cardiac involvement was in 1901, at the age of forty-seven. This was a heart attack with irregularity of the pulse, which occurred after running 300–400 yards. In his own description of this attack (Mackenzie, 1925) he notes that he " was conscious of a slight fluttering sensation, but suffered no distress of any kind." The pulse rate during this attack, which lasted two hours, was 90 per minute. A tracing, taken by himself, showed auricular fibrillation. During the next four or five years several attacks of this kind occurred, mostly after a full meal or when walking up a hill ; they lasted from ten minutes to half an hour and never caused any distress or limitation in his powers of walking.

The earliest symptom of limitation of effort was noticed by himself in 1907, and was represented by a slight feeling of constriction, hardly amounting to pain, in the upper part of the chest on severe continued exertion, and which soon ceased with rest. There were long periods when it was not experienced at all. For two years he was conscious of slight pain on effort under certain conditions, such as walking after a full meal or on a cold day. This pain he described as preceded by a sense of tightness or constriction, such as used to pull him up when running a race in boyhood.

In 1908, at the age of 55, Mackenzie experienced his first severe attack of cardiac pain. It occurred at night when resting, and followed a period of dining out at frequent intervals. The pain was across the chest and down the left arm ; it lasted two hours and varied in severity. Mackenzie further notes that in this attack " he could not be still but had to move about." After 10 grains of veronal sleep was obtained, and next day he was quite well and free from pain, though walking in the cold or after a meal still produced discomfort of an anginal type. This gradually became more noticeable, and by 1911 there was definite limitation of effort, though pain could be avoided by careful regulation of effort. From this time until the end of his life a somewhat anomalous symptom was present, to which he often referred, namely, that while a sustained effort produced pain, a sudden effort produced breathlessness without pain.

Mackenzie came to St. Andrews in 1918, and at that time was able to walk

at any pace from his home to the Cottage Hospital without discomfort, a distance of two miles. In 1919, when I first examined him, he was still able to do this and could play a round of golf regularly. The heart was then 1/4 inch external to the mid-clavicular line, the sounds were closed and well spaced, and, except for an occasional extrasystole, the rhythm was regular. Blood pressure was 156/92 mm. At this time he was also affected with intermittent claudication on continuous walking. He had first noticed this ten years previously after a rapid four-mile walk, but in 1919 it was evident after a short half-mile walk. The posterior tibial pulse was well felt on both sides. During the next five years this symptom was much less pronounced owing to the fact that pain in the chest occurred in response to a smaller effort than was necessary to produce claudication.

In 1922 limitation of effort prevented his playing golf, and even walking became difficult though by careful regulation of effort, severe pain was, in the main, avoided. A few very severe attacks occurred, like that in 1908, while resting. The most severe of all, in 1923, happened while he was sitting in his study in the afternoon, and lasted nearly an hour ; it was little influenced by nitroglycerine and was followed by extreme exhaustion. In August 1924 Mackenzie returned to London, and by this time only the gentlest of exercise was possible. Death followed a very severe and prolonged anginal attack in January 1925. As has been already mentioned, Dr. John Parkinson saw him shortly before his death, and has supplied the following note :

" On January 24 and 25, 1925, he suffered severe and prolonged attacks of anginal pain, and Dr. C. M. Anderson was called during the night. At 4.30 a.m. on January 25 he had morphine subcutaneously, gr.$\frac{1}{2}$, and chloroform inhalation for about an hour. It was necessary to repeat both at 8.20 p.m. on that day. At this time the pulse was 100 and regular and there was Cheyne-Stokes breathing. I did not myself see him until 10.30 p.m. that night (January 25), and he was then asleep. About 1 a.m. on January 26 he awoke free from pain and perfectly conscious and composed. He conversed cheerfully with Lady Mackenzie and me for a few minutes and then said he felt sleepy and soon he slept. At 4 a.m. his breathing changed and became irregular with long pauses, and a few minutes later the pulse stopped. There were no indications of pain at the end."

## EXAMINATION OF THE HEART

The heart was uniformly enlarged. Its weight was 18 oz. (510 g.) and it measured 14 cm. transversely, 13 cm. vertically and 28·5 cm. in circumference at the base of the ventricles. It should be mentioned that Mackenzie was a tall man of powerful physique, in whom a heart above the average size would be expected.

There was a considerable amount of sub-epicardial fat, especially in the right A-V sulcus, along the right border, and in the anterior interventricular

furrow. The epicardium was smooth and nowhere thickened. The veins on the surface of the heart were distended and more prominent than usual. On the anterior surface, numerous small vessels passed from the inter-ventricular furrow towards the left border, anastomosing with small vessels there, and these in turn with branches of a medium sized vessel on the inferior surface.

*The right auricle*, was dilated. Its wall was thin and, between the bundles of the pectinate muscles, translucent. The opening of the coronary sinus was large. The crista terminalis and the valve of the inferior vena cava were prominent. The tricuspid orifice and valve were of normal size and showed no pathological changes.

*Microscopical Examination.*—In longitudinal section the wall measured 7·5 mm. in thickness, of which the outer 6 mm. was chiefly adipose tissue containing a few scattered muscle bundles showing atrophic changes supervening on previous hypertrophy. In the outer fatty layers several small non-medullated nerve bundled were present, together with a few groups of sympathetic nerve cells. In the inner part of the wall the muscle bundles were more closely packed, the individual fibres were hypertrophied, and some showed atrophy and degeneration with increase of fibrous stroma between the fibres.

In transverse section the appearances were of a similar nature and indicated a considerable degree of ischæmic atrophy and fibrosis of some duration. There was no evidence of recent infarction, and foci of inflammatory infiltration were absent throughout the sections.

*The right ventricle* was large, its muscular wall rather thin, pale in colour and in places almost yellowish. There was no evidence of fibrosis of the wall. At the base of the infundibular portion, where the inner surface was smooth, the muscular coat was 6 mm. in thickness, while about the middle of the cavity, where papillary muscles were present, it measured 16 mm., of which more than half was sub-epicardial fat. The pulmonary valve was normal in structure and competent.

*Microscopical examination of the papillary muscles.*

(*a*) The sections showed a portion of papillary muscle on transverse section with a central arterial twig accompanied by a small nerve bundle. At one side was an area of adipose tissue separated from the endocardium by thin fibrous layer at one side and by a few muscle bundles at the other. The fibres of the papillary muscle were hypertrophied, but there was no increase of fibrous tissue and no evidence of old or recent infarction.

(*b*) The longitudinal sections of papillary muscle showed essentially similar features : marked infiltration of adipose tissue, hypertrophy of the muscle bundles and, in one group of sections, a mild degree of ischæmic fibrosis towards the attached end of the papillary muscles.

The arterial twigs in the papillary muscle had slightly thickened walls but their lumina were not narrowed.

*The left auricle* measured 7 cm. in diameter, both transversely and vertically. Its wall was thin and measured only 2·5 to 3 mm. in thickness. The mitral orifice admitted two fingers and the valve was competent. The marginal cusp was

FIG. 1.—Section of the wall of the left ventricle near the apex, showing a recent small hæmorrhagic infarction, involving the deeper part of the muscle wall, covered by a nodular reddish-brown mass of clot, the size of a cherry-stone, projecting into the cavity of the ventricle.

short and its surface nodular, especially near the attached margin. It was incompletely divided into three portions. It measured 6 cm. along its base and 17 mm. from the base to the free margin. The aortic cusp was, as is usual, much larger, and measured 24 mm. from its attached to its free margin. It showed no evidence of pathological changes.

*The left ventricle* was a large and thick walled chamber. The muscular wall, for the most part thick and firm, was 27 mm. in thickness near the base. At the apex, as usual, it was thin, and only 3 mm. in diameter. In colour it was somewhat pale. In its substance were several small whitish patches of fibrous tissue, in size from a pin's head upwards. In the anterior wall, 30 mm. above the apex, there was a patch of fibrous tissue 8 by 3 mm. and another patch of similar structure and size lay in the substance of the posterior wall, about midway between apex and base. At the apex there was a smal lrecent hæmorrhagic infarction involving the deeper part of the muscle wall, covered by a nodular reddish brown mass of clot the size of a cherry stone which projected into the cavity of the ventricle (Fig. 1). Section through this and the adjacent wall showed that the nodular tissue extended into the substance of the muscular wall, which was here reduced to a narrow margin 3 mm. thick.

*Microscopical examination.*—The section comprised the whole thickness of the ventricular wall. The muscle fibres in the outer layers were greatly hypertrophied and the interstitial tissue was of about normal amount. In the inner half the fibres showed a much greater degree of ischæmic fibrosis and irregularly shaped fibrous scars were numerous, in which muscle fibres in all stages of atrophy were found. The section did not contain any large branch of the coronary arteries and the smaller branches included showed a little thickening of their muscular coats, but no noteworthy intimal change.

The section of the infarction showed a portion of the apex of the left ventricle, the endocardium being partly covered with thrombus both old and recent. The muscle fibres were in general much hypertrophied, but there was a rather severe patchy ischæmic fibrosis which had produced marked thinning of the ventricular wall at the apex. On the internal aspect there was severe subendocardial fibrosis with many muscle fibres undergoing atrophy and degeneration and over this area there was a mass of old thrombus, decolorized in the centre and showing an early stage of organization. The greater part of the thrombus, however, was of recent coralline type and was well preserved, being only loosely attached to the wall. Between the thrombus portions of the columnæ carneæ were seen to be the seat of ischæmic fibrosis with fatty and vacuolar degeneration of the surviving fibres.

The apical myocardium did not show any sharply defined infarctions, but just at the apex there were several patches of more intensely eosinophile fibres whose nuclei appeared to be pyknotic and degenerate. Between these fibres there was a considerable degree of leucocytic infiltration which extended into the overlying epicardial fat. The small blood vessels in the fat were dilated and engorged with blood. None showed any evidence of thrombosis. It is probable that the areas of altered staining reaction and leucocytic infiltration were in a condition of early infarction.

*The aorta* had been divided 6 to 7 cm. from its root. At the point of division the lumen was cylindrical, measuring 33–34 mm. The ascending aorta showed a bulging to the right side (the bulb of the aorta) by which the

diameter was increased to some 45 mm. The interior of the ascending aorta showed extensive yellow mottling in patches, some separate and some discrete, 3–4 mm. in diameter. Near the root the mottling formed an arborescent pattern. The surface of these mottled areas was slightly raised. In thickness the wall measured 3·5–4 mm., but in places it measured 6 mm. On the posterior wall of the interior of the aorta was a large yellowish raised patch beginning about 35 mm. from the root of the aorta and extending beyond the level at which the vessel had been cut. In this area there was very considerable thickening of subintimal tissue and the tunica intima readily separated off from the other coats. The root of the aorta showed comparatively little pathological change. There were small thin yellow patches of atheroma round the root of the right coronary artery and adjacent to the orifice of the left coronary artery, but the lumen of these vessels was not materially narrowed. Except for the large area mentioned, the wall of the aorta was pliable and showed no general pathological alteration, there being only slight subintimal atheroma.

Section of the patches on the wall showed atheromatous changes of the intimal and subintimal coats. External to the smooth endothelium of the intima was a firm, pale yellow layer some 3 mm. in thickness ; external to this the darker coloured and almost unchanged tunica media. There was little, if any, calcification in the subintimal thickenings.

*Microscopical Examination.*—The aortic wall was greatly thickened by a large atheromatous patch which measured 1·5 mm. in thickness. External to this the

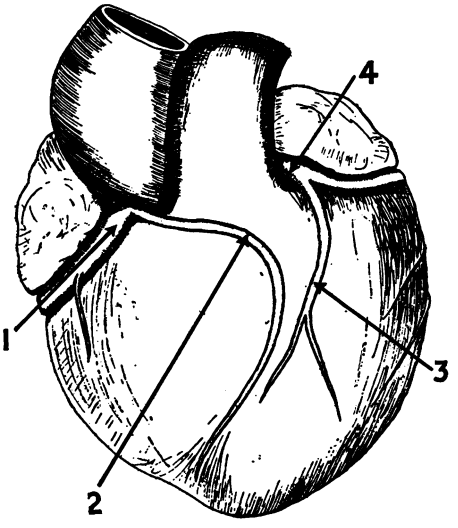


FIG. 2.—Arrangement of the coronary vessels on the anterior surface of heart.

(1) Stem of right coronary artery. (2) Interventricular branch of right coronary artery. (3) Interventricular branch of left coronary artery. (4) Left coronary artery.

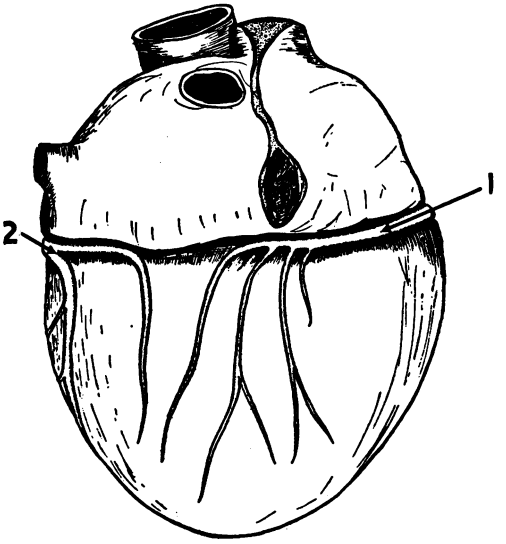


FIG. 3.—Arrangement of the coronary vessels on the inferior surface of heart.

(1) Right coronary artery. (2) Left coronary artery.

medial coat measured 1·15 mm. in thickness. The atheromatous lesion was of the usual type, consisting of degenerate fatty and granular material, with masses of foamy cells and also areas of clefts resulting from fatty crystals. In places the atheromatous lesion involved the innermost part of the media, with foamy cells between the musculo-elastic layers and degenerative changes in the inner elastic laminæ and muscle fibres. The middle coat was a little hypertrophied and in the outer part the tissue elements were comparatively healthy ; inflammatory cellular infiltration was absent and the elastic laminæ were intact.

*Coronary arteries.*—Both of the arteries and their branches were the seat of advanced and widespread degenerative changes, which had caused thickening of the wall of, especially, the medium-sized and smaller vessels, and diminution of their lumen. The arteries most affected were those in the anterior ventricular furrow, of which there were two, one from each coronary stem. These vessels were so thickened and calcified that their lumen was almost obliterated. A recently occluded vessel was not found as a cause for the infarction mentioned above.

*Right coronary artery.*—Near its root the external diameter of this vessel was 9 mm. Its wall was greatly thickened and the lumen, oval in outline, measured 2 by 3·6 mm. The thickening involved mainly the subintimal coat and also the tunica media, and the wall was firm and rigid. Section of the wall showed patches of degenerated cheesy material in the centre of the thickened areas, and the changes involved almost the whole circumference of the vessel.

From near the root of the artery a branch, 4 mm. in diameter, passed in the anterior interventricular sulcus to the inferior margin, lying by the side of a slightly larger branch from the stem of the left coronary artery. The two vessels ran side by side in the anterior longitudinal furrow, the left one giving a superficial branch and then entering the muscular coat half-way down, while the right artery ran onwards superficially. The wall of both of these vessels was greatly thickened, and the lumen in each reduced to a minute capillary cleft.

Half an inch from its root the diameter of the right coronary artery was 8 mm. At this point the thickening was less pronounced and the lumen wider. The artery continued as a large vessel and gave off numerous branches ; a small branch in the epicardial fat along the right margin ; a very tortuous branch which ran on the inferior surface an inch from the right margin ; a small vessel to the base of the ventricle ; at the left portion of the coronary sulcus three branches to the inferior surface of the ventricle, arising close to one another and measuring 2–3 mm. in diameter ; finally, the terminal portion of the artery turned downwards in the inferior interventricular sulcus. The distal portion of the artery showed much slighter pathological change, the lumen, though diminished, being distinct.

## Histological Examination

(*a*) *Stem of the right coronary artery* (Fig. 4).—The artery was greatly enlarged, the maximum diameter being 7·5 mm., owing to confluent patches of atheroma which surround the lumen and produced irregular narrowing. Adjacent to the lumen there was a thick subendothelial layer of hyaline connective tissue beneath which at one side there was a large crescentic patch of dense calcification, 3·5×2 mm., extending as far as the junction with the media, the muscular layer of which was thinned and atrophied. At the other side of the vessel the intima was fully 3 mm. in thickness and consisted chiefly of degenerate and granular material throughout which there are clefts left by dissolving out of fatty crystals. Foci of calcification were present in this material here and there. Red blood cells had seeped into the degenerate material in places and were seen in well preserved state in the superficial part of the atheromatous lesion. The medial coat of the artery was thin and atrophied. The adventitia had been largely stripped off during dissection. A small arterial twig seen leaving the parent vessel was practically unaffected by atheroma. There was no evidence of thrombosis in this portion.

(*b*) *More distal portion.*—This portion of the vessel measured 5 mm. in greatest diameter and was concentrically thickened, the lumen measuring 2 mm. The thickening was due chiefly to increase of the intima, which consisted of hyaline connective tissue with pronounced patchy granular and fatty degenerative changes in the deepest layers next to the media. These showed the usual cleft-like spaces where fatty crystals had been removed and there were several patches of calcification, the largest measuring 1·25 mm. in diameter. The media showed atrophy of the muscular layers which were reduced to a mere trace over the larger calcified patches. At the junction between media and intima there were, at several places, clusters of wide, thin-walled vessels filled with red cells and around these a mild round-celled infiltration. These appeared to be reactive, newly formed vascular channels, and from them a few thin-walled capillaries extended into the hyalinized layers of the thickened intima. The adventitial coat had been largely stripped off in dissection, but where present showed no evidence of inflammatory reaction. The lumen of the vessel was free from thrombus.

(*c*) *Ventricular branch.*—The vessel was smaller but showed similar though less severe change. There was again widespread atheroma, but the lumen was not so greatly reduced and there was an absence of the calcification so prominent at the higher level. The sections were not quite complete, a portion of the media being missing from one side in all sections. At one side the atheromatous intimal lesion consisted largely of foamy cells with patches of degeneration devoid of cellular structure, and at one point the surface had broken down and a small atheromatous ulcer had formed. This appeared to be very recent, as there were foamy cells mingled with fresh blood corpuscles in the lumen, but there was no evidence of thrombus formation over the damaged area. The tunica media was somewhat atrophied, but there was no noteworthy fibrosis. The adventitia showed no lesion.

(*d*) *Interventricular branch* (Fig. 5).—The artery was thickened, measuring 4×3 mm., and the lumen was eccentric and was reduced to a mere crescentic slit 1×0·3 mm. Opposite the convexity the intima was occupied by two large patches of very degenerate atheroma showing abundant fatty clefts and granular material. These patches had become confluent and both were surmounted by a mass of hyaline connective tissue. The larger patch showed much calcification of its outer portion next to the media. Opposite the convexity of the lumen the intima was thickened by multiple layers of hyaline stroma, without fatty degeneration in the deeper layers, and around the lumen the connective tissue was more loose and cellular and its arrangement suggested a recent formation indicative of arterial closure such as occurs in endarteritis from diminished blood flow.

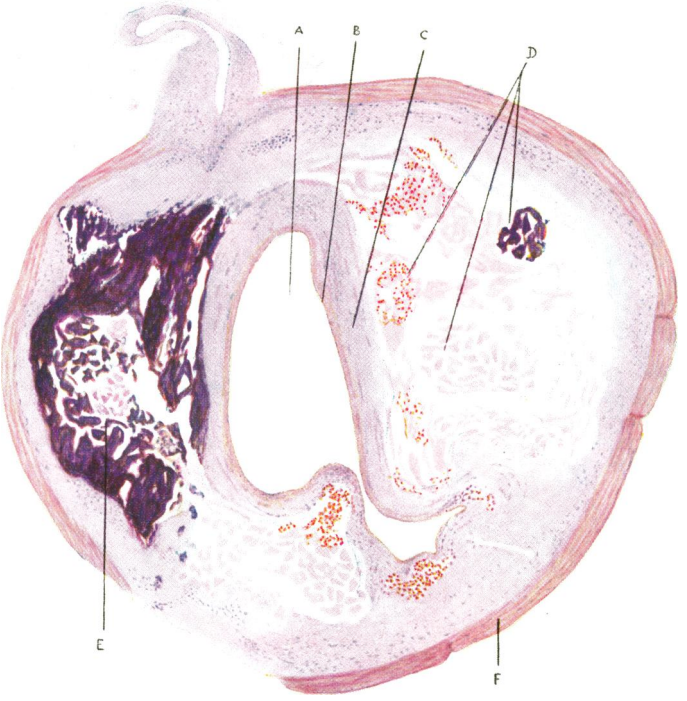


FIG. 4.—Section of the stem of the right coronary artery, showing the lumen irregularly narrowed by thickening of the sub-endothelial and middle layers, and patches of degeneration and calcification.

(A) Lumen. (B) Endothelial tunic. (C) Sub-endothelial tunic. (D) Patches of degeneration and calcification in middle tunic. (E) Large calcified area. (F) Adventitial tunic.

The media was much thinned and atrophied and overlying the larger calcified intimal patch there was complete disappearance of the muscular layer. A few wide, thin-walled vessels of newly-formed type were seen between the media and intima

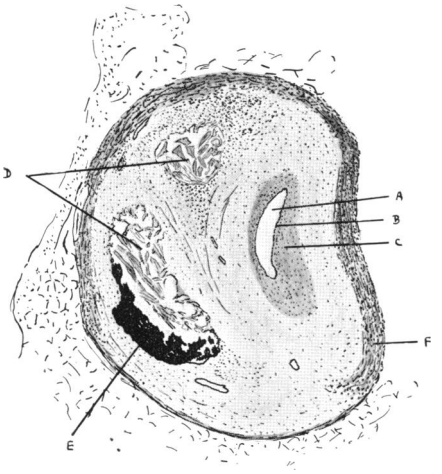


FIG. 5.—Section of interventricular branch of right coronary artery.
(A) Lumen. (B) Endothelial tunic. (C) Sub-endothelial tunic. (D) Patches of degeneration. (E) Calcified patch. (F) Adventitial tunic.

surrounded by round cells and a few foamy macrophages. In the adventitial coat there were several patches of round-cell infiltration, chiefly lymphocytes ; plasma cells did not appear to be present.

*The left coronary artery* was smaller than the right, and its wall was less affected by pathological change. The external diameter at the root was 6 mm. and the wall not more than 1 mm. thick. It gave off a large branch already mentioned to the anterior interventricular furrow ; the wall of this branch was more affected pathologically than the stem of the artery, there being marked intimal thickening in patches near the root, while an inch or two distally the thickening was even more pronounced, involving the whole wall and reducing the lumen to an extremely small size. The left coronary artery gave a large branch to the left margin of the heart. The wall of this branch too was distinctly thickened, and in the more distal portion so thickened as to reduce the lumen to the smallest dimensions.

(*a*) *Marginal branch* (Fig. 6).—The section showed a portion of cardiac muscle and one large artery and two small branches, the largest vessel measuring 3 mm. in diameter. The lumen was reduced to an eccentric elongated slit $1{\cdot}6 \times 0{\cdot}065$ mm. in diameter. At one side the intima was occupied by a large patch of degenerate atheroma consisting of granular material and clefts of fatty crystals with much calcification in the deeper layers. External to this area the media was almost totally destroyed and there was an abundant formation of new capillary vessels widely dilated and thin walled, between the deeper part of the intima and the media and at one point replacing the muscular wall completely. This newly-formed vascular tissue was

filled with round cells and macrophages, many of which were packed with dark brownish pigment which gave the Prussian blue reaction for iron. Similar cells were present in abundance in the adventitial coat overlying the lesion. The appearances suggested that there had been an interstitial hæmorrhage which had become organized

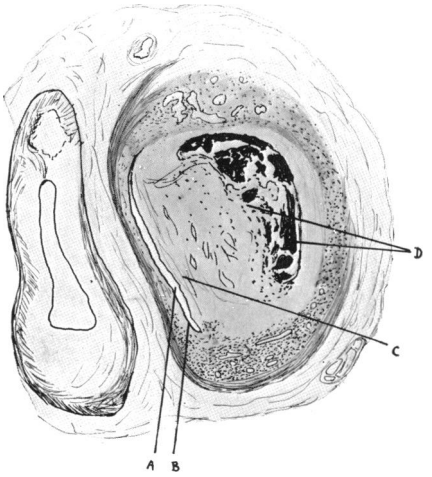


FIG. 6.—Section of marginal branch of left coronary artery, showing the diminished slit-like lumen and a large patch of degenerate atheroma, destruction of the media, and newly-formed vascular tissue replacing the muscular wall. Two smaller vessels show moderate sub-intimal thickening but absence of atheromatous disease.

(A) Lumen. (B) Endothelial tunic. (C) Sub-endothelial tunic. (D) Degenerated and calcified patches in middle tunic.

and repaired. The two smaller arterial branches showed a moderate degree of subendothelial intimal thickening, with musculo-elastic hyperplasia, but atheromatous disease was absent.

(*b*) *The interventricular branch* measured 3·2 mm. in diameter, the lumen, eccentric and almost circular, measuring 1·0 mm. The thickening was again due to severe atheroma, two patches showing marked degeneration and calcification being confluent, while less pronounced atheromatous deposits were also present on the opposite wall. The media was much thinned over the calcified patches, being almost devoid of muscular tissue.

There was also a minor degree of new capillary formation between media and intima and in several places thin walled capillaries had penetrated some distance into the degenerate intima and could be seen between the degenerate fatty patches and the subendothelial hyaline layer, which was, however, completely avascular.

The adventitial coat showed only a single small focus of round-cell infiltration and was devoid of pigment bearing macrophages.

### *Summary of Condition of Vessels*

Both coronary vessels and their branches showed extensive and far-advanced sclerosis in the form of patchy thickenings of the wall, which in some instances diminished and practically obliterated the lumen.

## THE CONDITION OF THE HEART IN RELATION TO THE SYMPTOMS

(1) The first signs of heart impairment occurred in 1908, seventeen years before his death, when he experienced a sharp attack of severe pain, which with our present knowledge would be diagnosed as due to a coronary thrombosis. There is evidence in the heart to confirm this view, for the patch of fibrosis near the apex corresponds to the structural damage which would be caused by such an attack.

Sir Thomas Lewis, who was good enough to send me his opinion after examining the heart, wrote to me as follows :

" Grant and I examined the heart very closely and we are agreed that there are amply sufficient old-standing changes at the apex of the heart to account for the first attack of pain described in his case notes. That attack of pain is strongly suggestive of coronary thrombosis, and the fibrosis at the apex is distributed in a way that also suggests thrombotic obstruction of an apical branch."

(2) The severe atheroma of the coronary arteries and their branches, with diminution of the lumen, affords ample cause for the occurrence of attacks of cardiac pain. Both of the arteries were affected and the anterior interventricular branch of each was greatly narrowed.

(3) There were numerous small patches of cicatrization in the substance of the muscular wall of the left ventricle. These patches though smaller were of the same nature as the larger fibrous patch at the apex, which was due to thrombosis of an apical branch. Other arteries to the left ventricle were profoundly altered and their lumen narrowed. This has been shown for example in the marginal artery of the left ventricle. There would therefore appear to have been numerous small thromboses at different times, each of which would be accompanied by symptoms similar to those experienced at the first attack. Several such attacks are recorded and the similarity is brought out in the case history. It is noted, for example, that on many occasions the attacks came on during rest and were quite unrelated to effort.

(4) The presence of numerous small blood vessels on the surface of the heart points to there having been an opening up of small vessels and the establishment of at least a partial anastomotic pathway for the supply of blood to the areas most severely impaired by the attacks of thrombosis. In this connection it may be remarked that during the last few months of his life between August 1924 and January 1925, Sir James's condition showed slight improvement. I found, for example, that not only could he walk for some distance down Exhibition Road from his home in Albert Hall Mansions, but he was able to walk up that road without distress, though at a slow pace. This improvement doubtless was due to a slight improvement, by anastomosis, in the arterial supply to the heart.

(5) The terminal severe attack of pain and cardiac impairment was associated with the occurrence of the recent infarction found at the apex of the left ventricle.

(6) There was no evidence of any impairment in the valves or in the genetic

system of the heart. The impairment was entirely in the muscular wall, brought about by the atheromatous disease of the arteries.

We have to thank Miss M. H. Kidston for preparing illustrations 1–6.

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