THE EVALUATION

of the

CLINICAL & ELECTROCARDIOGRAPHIC FINDINGS

in CORONARY INSUFFICIENCY.

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In any consideration of coronary insufficiency it is only natural that we should turn briefly to the early history of the study of the coronary arteries and coronary disease. The first clear description of the coronary arteries as such, seems to be found in a work by Harvey, "De Motu Cordis", first published in 1628 and here, although Harvey was able to describe the vessels or canals he had not recognised their function in supplying the nutrition of the heart muscle. It is to Heberden[1] however, that we have to turn for the first clear concise description of the group of clinical symptoms associated with coronary disease. In his book "The Cause and Cure of Disease" published about 1789 he devotes a chapter to this condition under the heading "Pector Dolis". In considering his observations we have to remind ourselves that they were made nearly 200 years ago and yet for clarity of description they comprise an amazing publication. We might even remark that in some respects little advance has been made in clinical observation to-day, although our scope for investigation both by the electrocardiograph and X Ray has been considerably widened.

Heberden had noted the relationship between exertion and the onset of pain "particularly if uphill" and even the relationship of the pain to food. He had also observed the disappearance of the pain with rest and its

substernal and often epigastric character with radiation to the arms. He had even observed how the pulse rate was seldom increased during the attack, and had observed aberrant forms of radiation such as into the right arm. What of course he was unable to recognise was the onset of coronary occlusion and his description of how ultimately all "suddenly fall down and perish almost immediately" was to him only some severe form of this same malady. He had noted even the extreme length and duration of the symptoms in some cases and the association with emotion, but he makes no final attempt to explain the condition although an aortic cause is suggested to him by Jenner, the pathologist.

It seems that the relationship of coronary artery disease to the pain was discussed by Jenner and Hillier at about this time, and it was Jenner who found ossified coronary arteries in his great friend Hunter who had died during an attack of angina, and who had suffered from angina pectoris during his lifetime.

As early as 1799 Parry is found to give a clear description of the coronary arteries as a cause of myocardial ischaemia, and a little later Allan Burns,(2) the Glasgow anatomist, in 1809 in a publication on disease of the heart adopted the coronary artery theory of Parry. (Levy).

In Europe about the middle of the nineteenth century "Herzchwiele" or myocardial fibrosis was being freely accepted as a common lesion particularly in Germany (Conheim, Weigert and Zeigler) and this theory was later revived by Mackenzie.

In France the end results of coronary occlusion were being set forth in 1896 by Mr. Réne Mare in a publication "L'Infarctus de Myocardie et ses consequances" and we have to wait for a clearer conception of these conditions until the discovery of the electrocardiograph by Einthoven in 1911.

Mackenzie,(3) in his "Principles of Diagnosis & Treatment in Heart Disease", alludes to the exhaustion of muscle from insufficiency of blood supply as the possible cause of the pain. He had noted the central origin of the pain and attributed the sense of suffocation to a spasm of the muscles of the chest wall.

It was not however till 1911 when Lewis published his "Clinical Disorders" and later still in 1915 on the publication of his book "Clinical Electrocardiography" that the investigation of this condition could be said to be on a sound basis.

Since the days of Heberden much work has been done on this subject but we owe it to Heberden for having put the clinical data in its first collective form.

In consideration of this condition we have to admit that the differential diagnosis of cardiac pain can be one

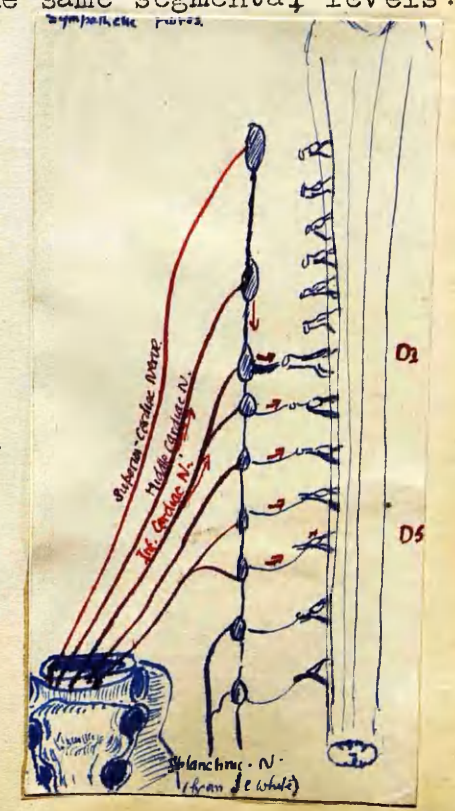
of the most difficult we are likely to encounter in the whole practice of medicine.

PATHWAYS OF CARDIAC SENSIBILITY.

Before we commence any consideration of Coronary conditions it is only natural that we should regard first of all the mechanism whereby pain of cardiac origin is conveyed to our consciousness.

INNERVATION.

In the cardioaortic innervation we may say that we have two separate nervous systems viz:- the vagal and the sympathetic, both of which come into close contact at various parts of their course, and in some parts actually anastomose. A further anastomosis occurs between the sympathetic fibres and certain somatic nerve fibres from corresponding dermatomes entering the same segmental levels. Both systems, vagal and sympathetic, are developed into a vast network of fibres of perivascular distribution and accompany these blood vessels to practically the whole head, neck, thorax and abdomen. At special localities and specified points each system develops well defined nerve strands, ganglia, and plexuses of nerves with frequent anastomotic branches between the two systems.

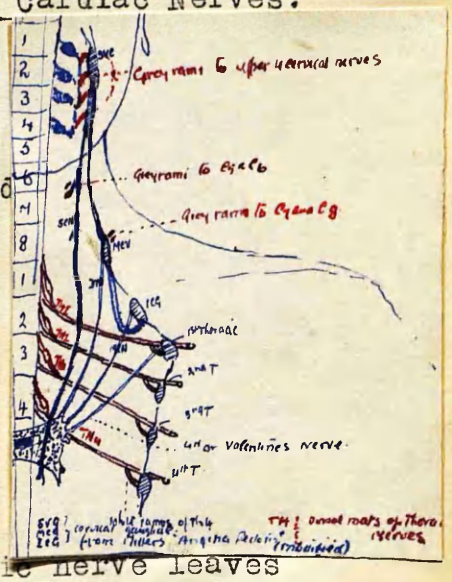


The Cervical Trunk and Ganglia.

The upper part of the sympathetic supply to the heart comes off a bilateral longitudinal tract which has several such ganglia along its length on each side and lies behind the carotid sheath.

The thoracic ganglia are ten in number, but only the upper four or five are concerned in the distribution of cardiac pain. These ganglia are united to the corresponding spinal nerves on each side. Each nerve has a visceral branch or white ramus joining it to the corresponding sympathetic ganglion, and a link of connection exists between the first and second thoracic nerves.

The Superior and Inferior Cardiac Nerves.



These act as strands for transmission of impulses between the cervical sympathetic ganglia, and the cardiac plexus. The superior nerve on each side anastomoses with the superior cardiac branch of the vagus and with both laryngeal nerves.

The inferior or small sympathetic nerve leaves the inferior cervical ganglion on the left side and passes behind the aorta but on the right it passes behind the innominate artery anastomising on each side with the recurrent laryngeal nerve. Another nerve sometimes present

is the fourth accessory sympathetic cardiac nerve or the "nerve of Valentine".

"White Rami".

Four or five white rami join the corresponding thoracic ganglia to corresponding segments in the spinal cord and these carry afferent fibres and efferent fibres which enter the spinal root ganglia of the posterior division of each thoracic nerve.

An interesting fact is that no white rami exist above the level of the 1st thoracic sympathetic ganglia, therefore all impulses above this must first descend to this level before crossing into the cord.

A more recently discovered set of grey rami on each side travels from the cardiac and pulmonary plexus back through the posterior mediastinum to the 2nd, 3rd and 4th and occasionally 5th & 6th thoracic sympathetic ganglia.

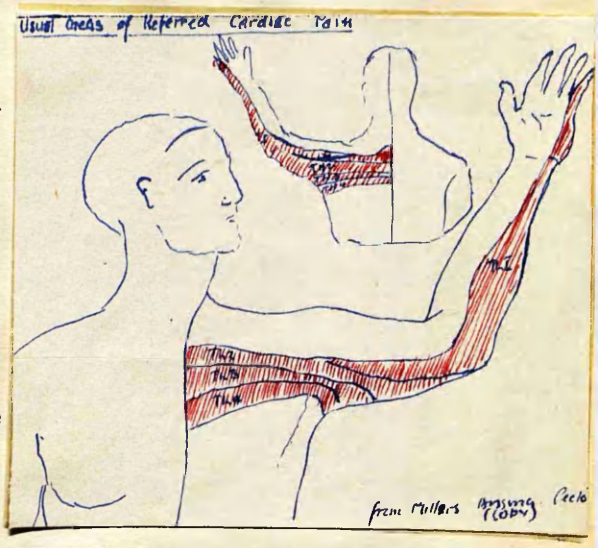
Efferent Sympathetic.

Of the efferent sympathetic little is known except that impulses pass via white rami to ganglia in the sympathetic chain and from this chain new groups are formed communicating with peripheral structures and visceral structures (heart).

In the study of referred cardiac pain we find that visceral afferent fibres from the heart and aorta are brought into relationship with somatic afferent fibres at the common entry i.e., the upper thoracic levels as a rule

TH1 - TH4 on the left side. These are the usual dermatones involved in cardiac pain, but occasionally C8 and TH5 are involved. In addition various uncommon districts are also liable to be associated with the pain and these will be described later.

The area of anterior dermatomic segments from C3 to TH6 are shown in red and these one might regard as the usual areas of referred anginal pain. The posterior aspect shows a similar arrangement. These illustrations show the usual areas of cardiac pain which is, as a general rule left sided. They cover the cutaneous surface of the posterior region and are continuous with the ventral aspect of the arm down to the web between 4th & 5th fingers including the ventral area of the left finger. Referred anginal pain stays within this boundary but there are notable exceptions. One very interesting fact is that on very few occasions does one find the posterior aspect complained of as the seat of pain.



The usual areas are frequently the upper zone supplied by the brachial plexus and upper cervical nerves C2, C3, C4 and C5, involving the surface of the upper chest and shoulders and C6, C7, C8 involving the ventral outer aspect of the arm.

The vagus consists of a large and extensive system of which the cardioaortic fibres are only a part. It seems to be undecided whether the vagus nerve really does transmit pain from the heart, and its chief function appears to be that of supervision and regulation of cardiac activity, including the lungs and gastro intestinal tract. It has been shown by surgery that severing of the sympathetic nerve alone can be sufficient to abolish the pain in some cases of angina.

The vagal innervation enters the cardioaortic plexus and also connects with many other plexuses in the thorax. The sympathetic and vagal systems have numerous interconnections and both systems are linked to many networks in the thorax, neck and head, and in fact impulses from them may be registered in almost any noncardiac plexus. We may therefore assume that cardiac impulses are brought into relationship with oesophageal, laryngeal, pharyngeal thyroid and pulmonary plexus; not to mention the brain itself. Anastomosis also occurs between the recurrent laryngeal nerve and the ansa loop and between the superior

cervical ganglion and the jugular and nodosal ganglion of vagus. When we consider the complexity of relations existing between the cardiac plexus of nerves and the sympathetic and vagal system with their widespread distribution; it is not difficult to understand the magnitude of the upset resulting in some cases from anginal seizures. When we consider the relationship to different nerve plexus of spinal origin it is easier to understand why the pain tends to appear in certain defined areas although the aberrant distribution of pain is not always easy to understand.

Coronary Arteries.

The right and left coronary arteries arise from the aortic sinuses immediately above the margin of the aortic semilunar valves.

Left Coronary Artery.

The left coronary artery shortly after its origin gives off the circumflex branch and then proceeds as the anterior descending artery. This last named artery courses down the anterior interventricular sulous to the apex around which it passes into the posterior interventricular sulous and extends upwards from a fourth to a third of the distance to the base of the ventricles. Occlusion of the branch vessels means infarction involving a varying amount of the anterior portion of the left ventricle and adjacent interventricular septum, the apex, and such portions of the posterior apical aspect of the left ventricle as are supplied by this artery.

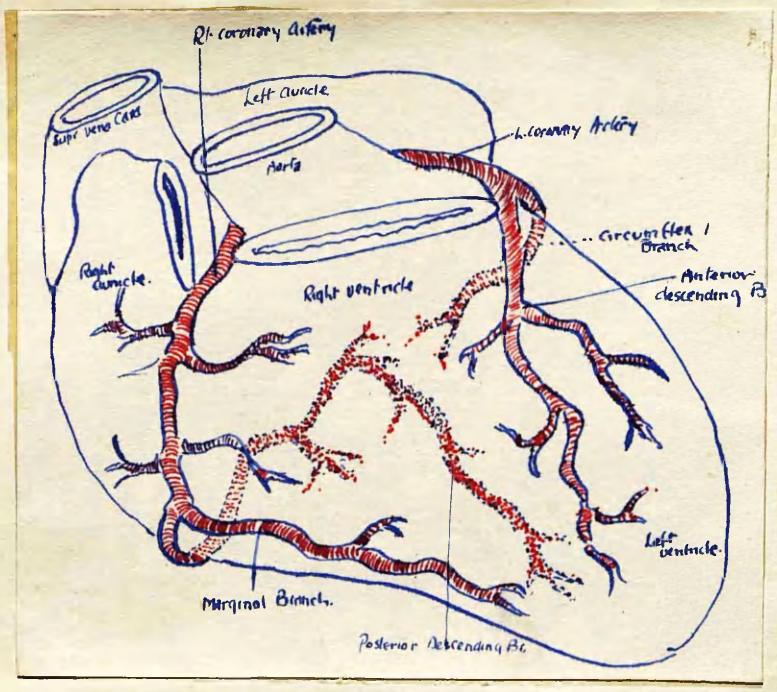
The Circumflex Branch.

This branch follows the coronary sulous for a variable distance turning off to supply the left third or half of the posterior portion of the basal 3/5ths of the left ventricle.

The Right Coronary Artery.

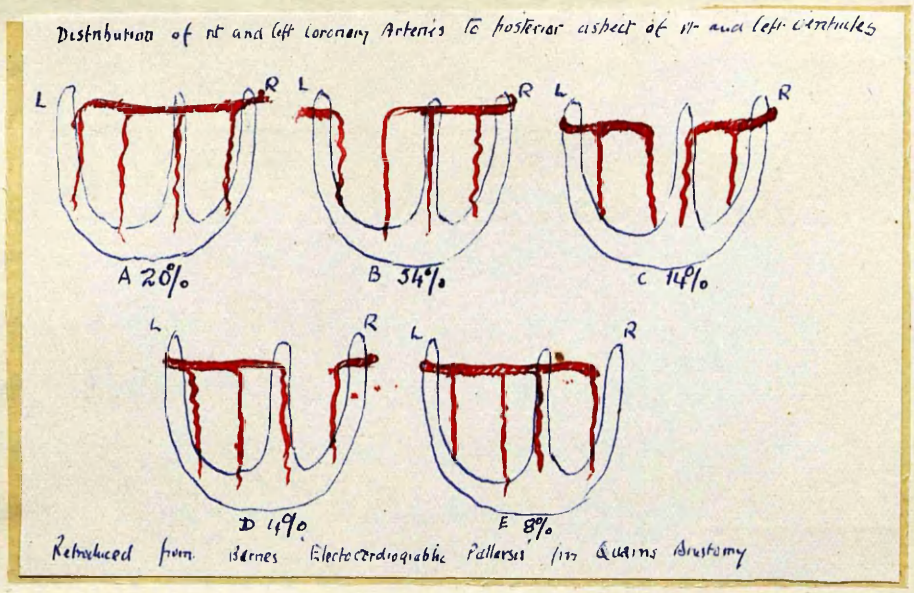
This artery runs into the coronary sulous until it reaches the posterior interventricular surface. Here it gives off a large branch - the posterior descending branch penetrating the interventricular septum. Generally the right coronary artery continues in the coronary sulous to reach the posterior surface of the left ventricle. Here it divides into two or more branches which course towards the apex but do not reach it. The coronary circulation is subject to a great number of variations in distribution, mostly involving the circumflex branch of the left coronary and the right coronary and sometimes even both arteries may arise from a common trunk in the aorta. These anomalies can and do play a tragic role in the outcome of acute coronary occlusion (Barnes)[4] and occlusion of one of these vessels supplying a large area of heart muscle results in a

large infarct which may be fatal.



By far the most important of these anomalies concerns the blood supply to the posterior aspect of the left ventricle. The circumflex branch of the left coronary artery may extend to supply all of the posterior and basal portion of the left ventricle including the posterior interventricular septum. The right coronary artery on the other hand may exceed its usual distribution and reach beyond the margin of the left ventricle as well as supplying the apex of the ventricles. In such cases it can easily be seen how extensive would be an occlusion of one or other of these abnormal vessels. Campbell in Quain's

Anatomy has put the frequency of distribution of these vessels in diagrammatic form which is worth reproducing.



A. in 20% right coronary supplies all posterior left ventricle.

B. in 54% right coronary supplies only posterior part l ventricle.

C. in 14% right coronary stops at posterior septum

D. in 4% circumflex br. of left coronary supplies all posterior left ventricle and posterior part of septum.

E. in 8% circumflex supplies most of left and right ventricles (posterior)

CORONARY INSUFFICIENCY.

Coronary insufficiency implies some degree of impairment of coronary supply and although in most cases an organic background exists, it can also be purely functional.

In the discussion of the causation of this condition, it is useful to bear in mind the enormous duty imposed on the coronary system even in health. The amazing efficiency of the cardiac pump and the still more amazing ability of the coronary system to supply the oxygen requirements of the heart at such extremely varying rates, makes us realise to what extent all exertion is limited by the efficiency of the coronary flow. Bramwell[5] states that one litre of blood will carry about 200 C.C. of oxygen and by the analysis of the arterial and venous blood it can be demonstrated that at rest the tissues will extract 35% of the oxygen passing through them. Since at rest the oxygen consumption of the body is about 250 C.C. per minute, the output of the left ventricle must be about four litres per minute. During severe exertion states Bramwell, the oxygen consumption amounts to 4.4 litres per minute, that is nearly 18 times the resting requirement.

Under such conditions the dilatation of the capillaries will perhaps allow a greater increased utilization of oxygen but this might be counteracted

by increased circulation of the blood in the lungs with consequent lowering of the oxygen saturation. If we include the right ventricle also we find that on exertion the total cardiac output reaches the enormous figure of 68 litres of blood per minute. The work done by the heart may be reckoned as the product of this output and the blood pressure.

Allowing for a 20% efficiency we may calculate the amount of oxygen required by the heart alone for this amount of work. We find that during severe exercise the heart muscle alone may consume as much oxygen as the remainder of the body at rest. Even the highly trained and physically fit athlete is therefore entirely limited in the extent of his exertion by the efficiency of his coronary circulation and it is not at all difficult to appreciate what little interference with this coronary system would be required to induce symptoms of myocardial ischaemia particularly when the heart is at work under load.

Aortic Theory.

It is easily shewn by reference to the literature of the past, that the coronary arteries or the result of impairment thereof has not always been accepted as the prime cause of anginal pain.

Heberden's original theory hinted at the possibility of an aortic origin of this pain, and certain cases of anginal pain have shewn aortitis at autopsy, but it does seem that in most cases of aortitis e.g. syphilitic aortitis pain is seldom experienced unless the openings of the coronary arteries are actually involved in an inflammatory process or unless definite aortic regurgitation with a subsequent low diastolic pressure is present. Albutt[6] maintained that the pain was due to distension of the first part of the aorta with subsequent stretching of the nerve endings in the aortic wall. Findings at autopsy have shown great discrepancies in this aortic theory and there seems little doubt that aortic lesions generally have been found to be unassociated with pain during the patient's lifetime. In arterio sclerosis and rheumatic disease involving the aorta, pain is not usually a prominent feature, although in many cases arterio sclerosis is found where a history of anginal pain is obtained.

Lewis[7] discusses experiments on animals in his recent book "Pain" and demonstrates how puncturing of the aorta in dogs appears to be painless. There does appear however to be some proof that distension of the aorta can produce pain, and this theory of causation still has some adherents. We must bear in mind the

pain of aneurism of the aorta although probably most of the symptoms here are due to pressure.

Coronary Spasm.

Coronary Spasm might be described as a second theory of causation, and it does seem that there are many good reasons for the association of the pain with spasm of the coronary arteries, even if it be only accepted as one of the initiating factors in the condition. Lewis gives an illustration of experiments conducted on the animal and shows that traction on the coronary artery can produce pain and signs of limping of the left leg in the dog, but that ligation of the coronary artery appears to be painless. In this same treatise Lewis goes to considerable length to discuss a series of experiments conducted in conjunction with Pickering & Rothschild on this same subject. Here he maintains that the spasm occurs after the onset of pain and not during it. A comparison is drawn between the spasm of the coronary arteries and the spasm of peripheral arterioles in Raynaud's disease or in Intermittent Claudication. Lewis maintains that in Raynaud's disease it is only where nutritional changes or focal neurosis have occurred that pain is experienced, and that this pain is confined to the area of the necrosis only. It is quite conceivable that such focal necrosis occurs in heart muscle also, and that some

of the pain is located in such areas of muscle damage when these areas are deprived of normal blood supply. It has been suggested by some authorities on the other side of the Atlantic that coronary spasm of any marked degree is extremely unlikely in vessels which are in all probability, the seat of extensive solerosis and that the coronary artery is so limited in its supply of muscle that such a spasm would not be great at any time.

There does not appear to be universally much support for this statement, and in fact, it is all rather negatived by observations on a case of Intermittent Claudication. During such an attack the Dorsalis Pedis artery can actually be seen to contract in the foot, although in some cases it may even be the seat of advanced arterio solerosis.

Dilatation of the Coronary Artery.

Another theory put forward by some is that the pain is due to dilatation of the coronary artery proximal to the spasm, and there seems to be little doubt that this is a factor, and it has been shown that during animal experiments the coronary arteries can actually be seen to dilate. Of the two latter theories the consensus of opinion appears to be that both spasm and dilatation of the affected coronaries are at least, in some small way, associated with the initiation of the pain.

In many cases of coronary insufficiency it is not

uncommon, to come across examples of extreme vasomotor sensitivity associated with symptoms of coronary insufficiency, and one is always inclined to suspect that it is at least a predisposing cause. Such wide variations of vasomotor sensitivity in the form of migraine, hemianopias of various types; numbness of the hands and feet are frequently associated with anginal pain, and one always makes a special enquiry as to the existence of such symptoms in all cases suggestive of anginal pain. It has always seemed to me that it would be worth while to conduct a series of cold pressure tests on these cases of simple angina of effort, and I have no doubt that the results would shew that a high percentage of cases have the very sensitive vasomotor systems suggestive of The Hypertensive Diathesis.

It is shewn very conclusively by Lewis in his book "Pain" where he describes his experiments conducted with the help of Pickering & Rothschild, that spasm of the coronary artery can only be an initiating factor.

Here he conducts experiments chiefly on skeletal muscle and the results are so ably tabulated as to leave little doubt in our minds that the cause of the pain is to be found in the muscle itself. Lewis points out that the spasm of the arterioles is not directly related to the pain, since by restricting the blood flow to the muscle

and releasing it again, he found that the rate of blood flow into the restricted limb during the first few seconds of release, was greatly in excess of the normal. Since at this precise moment, pain was being experienced, it was obvious that the pain was not associated with constriction of the arteries.

In this same chapter Lewis goes on to describe what one might call his epoch making experiments by restricting the blood flow to skeletal muscle at work. He demonstrates very clearly that muscle working in a restricted blood supply develops pain in 24-43 seconds, and becomes too disagreeable for the conduct of exercise in from 60-80 seconds. These figures are found to be surprisingly constant, and one important fact emerges, that the pain, in common with the pain of coronary insufficiency was continuous i.e. it did not come and go, and no cramp or stiffening of the muscle was observed. On release of the circulation the pain vanished in about 3 seconds. Another important point brought to light by these experiments was, that if the exercise and restriction were continued the pain persisted at about the same level until the circulation was restored. This led to the natural assumption that the pain was due to some substance either of a chemical or physiological nature within the muscle

and formed when the muscle was working in anaerobic conditions and only removable or changeable by the blood flow.

To suspect lack of oxygen as the prime cause of the condition was the natural and logical step following these findings. We come therefore to what is now regarded; chiefly on the basis of Lewis', work as the cause of pain in coronary insufficiency, namely lack of oxygen supply to the heart muscle and certain changes occurring within the heart muscle as the result of this deficiency. When that muscle is working in a limited oxygen supply Lewis shows us clearly in subsequent experiments that this oxygen lack, "per se", is not sufficient to induce pain in the limb, if it be at rest; and in fact continuous arrest of the circulation even to the point of cyanosis (if the limb be kept warm and at rest) is not enough to produce the pain.

Therefore Lewis concludes that this pain must be due to some process starting within the muscle and connected with its contraction, but not excluding the idea of oxygen want as a promoting factor. We know that pain can develop in muscle well supplied with blood, if that blood be deficient in haemoglobin, such as in anaemia or if that blood supply be deficient in oxygen. Lewis came to the conclusion therefore, that this substance is formed within

the muscle fibre and must diffuse out to the tissue spaces, and thus act on the nerve endings situated there. This substance Lewis has called the "P" or pain producing substance. It is obviously formed during contraction of muscle and must gradually rise in quantity until the threshold for pain for that particular individual is reached. From this point it still increases with exercise and can be altered or dispersed with the returning blood flow in 3 seconds. Factor P can develop in normal muscle with adequate blood supply, but does not rise above the pain threshold. Lewis shows that a latent period occurs between the occlusion and the appearance of the pain, during which he assumes the P substance to be rising in the tissue spaces to the level corresponding to the level in the muscle fibres. He puts forward the suggestion that the reason for the absence of pain where there is no arrest of circulation may be due to an adequate interchange between the tissue space and oxygenated blood in the vessels.

In applying these findings to cardiac muscle it is not difficult to see where even a slight restriction of the blood supply to the myocardium would very readily induce pain in an organ which is so constantly functioning although the vascularity of heart muscle is considerable. We know that coronary insufficiency can be induced in the normal healthy individual by violent exertion of a

magnitude sufficient to produce a great discrepancy between the oxygen want of the cardiac muscle and the ability of the coronary circulation to cope with the demand. I think we should regard this P substance somewhat in the light of a protective or defensive mechanism against cardiac overstrain. We know it has been demonstrated that distensibility of and subsequent pain from the aorta is such a mechanism, but it seems to me that nature has provided a very efficient safeguard in the formation of this pain producing substance in cardiac muscle. Some close affinity exists between the P substance and lactic acid, and it is maintained that a constantly reversible process of lactic acid to glycogen takes place within the heart muscle.

Lewis follows up these findings by injecting lactic acid into muscle and is able to demonstrate the similarity of the resulting pain both in duration and character to the pain induced by ischaemia. In cardiac muscle any fibrosis or damage must be a factor in the limitation of exchange of oxygen between the muscle space and the circulation and it might conceivably be one of the reasons for the continuation of the pain or at least may be a factor in its earlier production.

If this ischaemia be too long continued myocardial infarction with damege to heart muscle can ensue.

Some authorities attempt to suggest that all cases of the "anginal syndrome" result from an initial infarction either recognised or unrecognised. This appears to be a wide sweeping statement which can be disputed but there seems little doubt that more recent methods of electrocardiographic investigation seem to suggest that this must be the case in 59 - 60% of cases. This is no doubt due to the extensive use of posterior chest leads, and these leads certainly have the advantage of being able to demonstrate posterior myocardial infarction with a higher percentage of accuracy than the classical leads and leads IVF or 1 VR alone. However, it does appear that certain cases of what one might call simple coronary insufficiency do exist as a clinical entity, distinct from cases resulting from coronary occlusion. This group seem to have a slightly less intense clinical history and the prognosis appears to be better.

In this investigation an attempt has been made to assess the relative value of the clinical, electrocardiograph and X Ray findings in this condition of coronary insufficiency and to try to isolate the dominant features of each source of information which can be of value as diagnostic aids.

It has been maintained by some authorities that where abnormal electrocardiographic findings appear the

prognosis is not so favourable and the clinical findings are more definite. This I have no doubt may be the case, but the great difficulty is to establish just what electro-cardiographic findings are acceptable as abnormal.

We have therefore established that the cause of the pain in coronary insufficiency is due to a disproportion in the supply of blood and oxygen to the heart muscle and the demand for oxygen on the part of the muscle itself. The two most important results of this are ischaemia and anoxia of the heart muscle. Local anoxia seems to be the accepted cause of pain and prolonged ischaemia may produce fibrosis, hypertrophy and subsequent congestive failure. The most usual cause of this restriction of coronary supply is arterio-sclerosis of the coronary arteries themselves. There are usually two coronary arteries, the right and left, leading directly from the aortic wall, and although a very limited collateral supply does exist, one is justified in classing these arteries as end arteries.

Some authorities believe that a certain amount of backward supply may occur through the Thebesian veins, but this does not appear to be generally accepted, and the heart muscle depends almost entirely on the coronary arteries for its blood supply. Therefore, any disease blocking the coronary arteries must occur in the length of the vessel or at its mouth. In the first condition

this narrowing may be a part of a general arteriosclerosis involving the whole of the arterial system, or it may be part of a local disease or atheroma involving the aorta alone, with obstruction of the coronary ostia or spread into the coronary vessels; as for example in syphilitic aortitis. The fact that the coronary vessels are frequently involved in such an accident, and of such a serious character, is an indication of the unusual stresses which these vessels are called upon to bear. The position of the coronary orifices, situated as they are just above the aortic ring exposes them momentarily to great stress during the closure of the aortic cusps. This pressure lasts for a very short time and is quickly relieved by the elasticity of the aorta, but of course it is constantly occurring.

We must also remember the extensive distribution of the coronary vessels throughout the cardiac musculature which is constantly undergoing contraction, and thus subjecting the vessels to alternate compression and relaxation. The main coronary vessels and sub-epicardial branches not subject to pressure are filled with blood under systolic pressure and cannot be emptied, but the principal coronary circulation occurs during diastole. It is hardly surprising that we find as the result of this extraordinary stress that these vessels can in some cases

develop a thickened intima in very early life (even in the teens) and what is more important that this may occur quite apart from the existence of arterio sclerosis elsewhere.

I believe that similar findings occur in arteries subject to similar stress in other parts of the body e.g. uterus, ovaries, intercostal and hepatic arteries but none shewing such extensive thickening as the coronary vessels. The left coronary and its descending branch appears to be most frequently involved in this type of lesion, and particularly the first 3 or 4 centimetres of its length where coronary occlusion is found to occur in the greatest number of cases, and it is significant that at this particular site the left artery gives off its main branch turning at right angles as it does so. The right artery peculiarly enough does not appear to have the same tendency to involvement in this way.
An article by Leary (8) is worth quoting:-

> "In my series, advanced coronary sclerosis at 12 and 15 years of age, and coronary deaths at 24 - 29 years of age, and early in the thirties determine the issue beyond question. Many of the victims were young, healthy athletic types with no stigma of senility."

As Lewis has pointed out where we have contraction on the part of the heart muscle in limited oxygen supply we have pain, and as chronic disease narrows the lumen of the coronary artery, we have a chronic myocarditis gradually resulting from recurrent attacks of myocardial

ischaemia. This condition is described by many writers as chronic or protracted coronary insufficiency. As the blood supply becomes more restricted we have gradual atrophy of the muscle fibres with replacement by scar tissue and this one might describe as chronic coronary insufficiency as distinct from the acute condition or coronary occlusion. This occurrence where a sudden cutting off of the blood supply before any collateral circulation can be established leads to acute ischaemia with subsequent necrosis of the muscle fibres and supporting connective tissue, followed by an inflammatory exudation. Midway between these two lesions must lie a condition which might be called petechial thrombosis with minute necrosis of muscle fibres in small local areas, but with stimulation of the supporting connecting tissue. This has been described by some authors as miliary infarction or miliary myocardosis - the former appears to be a very suitable description of this condition. It is always possible for a thrombus blocking the coronary artery to undergo canalization with re-establishment of the circulation, but as a rule, the circulation is re-established by the development of some collateral supply. In this connection it is interesting to note that some authorities maintain that the collateral circulation is much greater where the subperiocardial fat is great and hence that acute myocardial infarctions are less frequent

and less extensive in the aged adipose subjects.

It is maintained that athero-sclerosis as distinct from arterio-sclerosis is marked by the presence of foam cells in the subendothelial layer of the intima of the coronary vessels. This whole condition is now known to be due to excess of cholesterol and is assumed to be due to some error in cholesterol metabolism. This may be associated in some way with the reported frequency of occurrence of anginal symptoms and gall-bladder disease, since cholecystitis and angina are both liable to be confused one with the other, but are supposed by some authorities to occur together although this is still doubted by many.

The obstruction of the coronary vessels by an embolus or piece of vegetation from an aortic valve is not uncommon. Other conditions can produce an inadequate coronary flow apart from athero-sclerosis such as:-

1. Athero-sclerosis with calcification and narrowing
2. Syphilis of the aorta with stenosis of one or both coronary orifices
3. Emboli or thrombi from the mitral or aortic valve
4. Rheumatic fever with involvement of the coronary walls
5. Periarteritis nodosa with coronary involvement

Other causes of a systemic and general nature will be discussed under appropriate headings.

We must bear in mind that various clinical conditions apart from spasm or disease of the coronary artery can induce coronary insufficiency. We can roughly divide these into two separate groups:-

1. Cardiac conditions
& 2. Systemic or general conditions.

CARDIAC CONDITIONS.

Coronary Sclerosis.

Here we have naturally and primarily diseases of the coronary arteries themselves, such as arterio-sclerosis which may be limited or rapidly progressive. Where the condition is limited we have slowly progressive or sometimes stationary symptoms of coronary insufficiency or mild "angina of effort", in some cases continuing for many years without change or even no symptoms at all.

The rapidly progressive sclerosis producing calcification of the coronary artery is usually associated with a history of an angina of rapidly increasing frequency and severity. This condition is very much more prone to occlusive changes either as the result of the gradual silting up of the vessels by advanced sclerosis or due to the sudden formation of a thrombus with subsequent myocardial infarction.

Syphilis of Aorta.

Syphilis of the aorta leading to involvement and stenosis of the coronary arteries is usually confined to the first few millimetres of the aorta and is a frequent cause of coronary insufficiency.

Embolism.

Embolism of the coronaries following the impaction of vegetations separated from the mitral and aortic valves or obstruction of the coronary ostia by a thrombus covering a patch of sclerosis at the root of the aorta can likewise produce coronary obstruction.

Rheumatic Fever.

Rheumatic fever with involvement of the walls of the coronary arteries and subsequent thrombus formation therein is another source of coronary obstruction.

Aortic Diseases.

Aortic valvular disease especially stenosis is the common cause of coronary insufficiency producing as it does a diminished blood flow into the aorta and thence to the coronary artery. A similar result ensues in aortic regurgitation, and here we have a low diastolic pressure in the aorta with a diminished flow to the coronary vessels. Aortic regurgitation is a more frequent offender in this respect than aortic stenosis.

SYSTEMIC CAUSES.

Of these, one of the most important is anaemia, producing as it does a blood supply deficient in oxygen carrying capacity either as the result of a low haemoglobin or diminished red cell count. Of these two, lack of haemoglobin is by much more the usual factor in the causation of pain. If the coronary flow is somewhat decreased as the result of coronary sclerosis then the affect of anaemia is much more likely to be felt. In these circumstances the coronary system may be slightly damaged and function quite adequately under normal conditions, but may fail under adverse conditions, and as the result of successive demands following exercise. This is often seen following sudden haemorrhage in post operative conditions or is occasionally encountered in menorrhagias. I have in mind at the moment a patient with a typical history of angina or effort who has had attacks of substernal pain induced by giving blood as a donor. The haemoglobin percentage here was 90 red cell count five million, so that one can assume that her coronary sclerosis was made more obvious by a temporary fall in volume only.

We know that sudden haemorrhage can induce acute coronary insufficiency of sufficient severity to produce anginal pain and electrocardiographic changes even in the normal heart, and that these changes are of a transient

nature disappearing with treatment. This is equally true of pernicious anaemia, where a diminution in red cells more than diminution in haemoglobin is the cause, and I have seen many cases of substernal pain or pain suggestive of the anginal syndrome clear up with the appropriate treatment of liver injections etc. Microcytic anaemias associated say, with peptic ulcer, are equally responsible for similar onset of pain, and in fact, it is sometimes difficult to be sure whether one is dealing with the pain which is due to the ulcer or a pain of coronary insufficiency.

In older patients with peptic ulcers and haematemesis the danger is not only that coronary insufficiency may ensue, but that coronary occlusion may follow a haemorrhage since we are frequently dealing here with already sclerotic vessels and therefore probably coronary sclerosis of an advanced type. In the question of reduction of oxygen capacity of the blood we must remember various other factors, mostly of a toxic nature which can reduce or alter the oxygen carrying capacity of the circulatory system. Carbon monoxide poisoning producing as it does, the inert carboxy haemoglobin has been known to produce attacks of anginal pain. We have also the records of pain of this type induced by working in chemical fumes or in dusty atmospheres, and I am inclined to believe that in these cases the effect of the fumes may be more pronounced on the oxygen carrying capacity of the blood than any

effect on the myocardium.

It is suggested by some American writers that the changes produced in the electrocardiograph as the result of inhalation of tobacco smoke are the result of oxygen deprivation only and not due to any toxic effect of derivatives of the tobacco.

I have in mind at the moment one patient who dates the onset of her attacks of anginal pain on effort from her employment two years ago in a chemical factory where, I understand the fumes are mostly sulphur dioxide.

In this connection we must always remember anaesthesia as a cause of anoxia, and I understand that electrocardiographic records pursued mostly in the United States during anaesthesia have shown typical electrocardiographic changes associated with coronary insufficiency, and that anaesthetic deaths resulting in ventricular fibrillation are the direct result of this.

Another important and what one might call physiological factor influencing the amount of oxygen in the blood is the question of high altitude. Cardiac pain and electrocardiographic changes have been found to occur amongst mountaineers and pilots, and also amongst workers in mines, where of course, low oxygen intake is a predisposing factor inducing a myocardial ischaemia. We must not forget that a pain simulating the pain of angina pectoris can be induced in the normal healthy individual by violent exercise following

a full meal. No doubt, in this case, we have a drop in the coronary circulation due to the demand of the splanchnic vessels for a greater supply of blood for digestion. At the same time another factor may come into play, i.e. the pushing up of the diaphragm following the meal with subsequent rotation of the heart and possible reduction in the coronary circulation by rotation of the heart about its axis.

While discussing the question of food in relation to this type of pain, we must bear in mind the whole question of food allergy as a definite factor in causation. Several cases are quoted in the American Journals where apparently true anginal attacks have been completely cured by the avoidance of certain foodstuffs to which the patient was allergic. The effect of cold on the coronary circulation is well known and is usually regarded as an aggravating feature in all cases of coronary insufficiency.

A naval officer who has spent a good deal of time on various Arctic explorations tells me that it is a common occurrence for at least 20% of the ship's crew to suffer intense substernal pain on coming suddenly on deck from the warm ward room, particularly in the presence of icebergs. I understand that a method of gradual acclimatisation is employed, by spending a short time in a cold room before going on deck.

Other toxic factors such as arsenic and lead are often associated with this type of pain, and we can include in

this group, coffee, tea and adrenalin. The whole question of tobacco as a causative factor seems to be under debate, but there seems little doubt that certain individuals are sufficiently susceptible or allergic to some substance in tobacco smoke which can produce the pain simulating angina pectoris. I have not seen a definite case with true anginal symptoms clear up on cessation of tobacco, but I understand that one or two cases of pain simulating angina and resulting from tobacco have been found at the Heart Hospital, London. Experiments carried out in the United States and illustrated in Graybiel & White[9] shew a definite drop in the height of the T waves in Leads 1 and 11 after inhalation of tobacco smoke, and it is suggested here that the action may be due to parasympathetic paralysis similar to that found after atropin administration, or to diminished oxygen intake.

We must remember that alterations in cardiac rate are liable to produce diminished oxygen carrying capacity of the blood, and increased anoxia of part of the heart muscle, due to decreased diastolic filling with drop in stroke volume. We have thus established a relative deficiency in coronary flow, and this is especially so in Paroxysmal Tachycardia although rarely in simple tachycardia.

In hyperthyroidism we have this condition occurring fairly frequently, and some cases of anginal pain have been completely relieved on ablation of the thyroid. In the latter condition, no doubt, another factor is the increased

metabolic rate and greater demand on the part of the body for oxygen.

At the other end of the scale we have occasional cases occurring in a bradycardia or in the slow circulation of myxoedema, and I have one or two cases in my own records where pain similar to that of angina has cleared up on thyroid therapy. These details will be discussed more fully in the chapter on Differential Diagnosis.

CORONARY OCCLUSION or THROMBOSIS.

No consideration of coronary insufficiency is possible without discussing the whole question of coronary thrombosis with which it is closely allied, and in some cases inseparable. The recent work on this subject has furnished concrete evidence of the part played by the disorders of the coronary artery in the production of the pain of occlusion. Usually the clinical picture is dramatic in the extreme, and appears often like a "bolt from the blue" in middle aged persons who have never had any cardiac pain. It usually comes on after a large meal or sometimes after the patient has gone to bed. It will be more fully discussed in a later chapter, but it is important that it should be mentioned at this stage, since the differential diagnosis between it and coronary insufficiency is liable to crop up frequently during the course of our discussion. An interesting communication on this whole subject is to be found in the American Heart Journal, January 1942. Here Boas (10) raises the problem of whether myocardial infarction is merely a fortuitous occurrence or is preceded by symptoms of definite cardiac disease. He maintains that in cases where the development of arterial disease was comparatively slow there was time for the establishment of an adequate collateral circulation so that when one of the coronary arteries was occluded infarction of the myocardium did not take place.

Blumgart in the same article maintains that when patients who are previously well suddenly develop angina particularly at rest one must suspect coronary narrowing or occlusion. It is the conviction of the writer (Boas) that such patients who are ordinarily regarded as having angina have at the outset experienced organic damage to one or other of their coronary arteries leading either to permanent narrowing or occlusion of the vessel.

Blumgart and his associates called attention to cases of prolonged attacks of classical angina not followed by fever or any other evidence of infarction, and showing no post morten changes. These attacks he maintained are due to coronary failure producing an irreversible myocardial ischaemia coinciding with increased demands on the heart muscle, and provoked by abnormal rhythm, emotion or exertion, or insufficiency of flow following shock or haemorrhage. These writers raise the very interesting possibility of haemorrhage into the wall of the coronary vessels as a factor in the sudden production of coronary occlusion. This certainly appears to throw some light on the occurrence of these cases of myocardial infarction where no history of arteriosclerosis or hypertension is obtainable, and where no previous history suggestive of coronary insufficiency can be obtained. It is suggested that in some cases indirect trauma of the heart may also account for the onset

of coronary thrombosis and there does appear to be sufficient clinical data to support such conjecture. In fact the majority of cases of coronary occlusion are found in the absence of anything suggestive of previous coronary insufficiency and where no limitation of exercise tolerance can be found prior to the occlusion.

It is worth recalling that in our previous discussion it is mentioned that the coronary arteries can be the sole focus of athero sclerosis where no generalised arteriosclerosis or hypertension is manifest.

Boas discusses the possibility of a direct blow on the chest wall producing occlusion of the coronaries or injury sufficient to produce stenosis and supports this by several illustrations.

In summing up, we can only come to this conclusion that any upset of the balance between the nutritional needs of the heart muscle and the adequacy of the coronary blood flow, if sufficiently long continued or sufficiently complete, can produce myocardial infarction with subsequent necrosis.

CLINICAL ASPECTS OF ANGINAL PAIN.

The investigation of cases of anginal pain or the pain of coronary insufficiency is one of the great problems facing the practitioner. No condition can have quite so many alternative causes which may so closely simulate it, and no history can present a greater task in its elucidation.

For some reason difficult to assess, obtaining a clear history of anginal pain seems to be fraught with great difficulties, and can be a very considerable test of patience. In spite of all attempts to avoid it, we find ourselves compelled to resort to leading questions in an attempt to get any satisfactory and concise history.

We might say that in no condition can a leading question be more dangerous than in this sometimes complex malady, since most patients have frequent reference to current literature and books of a semi-medical nature, and are very prone to build up for themselves a clinical picture which requires little encouragement to distort. Many times it would often seem as if the patient were being deliberately vague and obtuse, but we must always bear in mind that even with the best will in the world the symptoms can be extremely variable in the mode of onset and character. It is not as a rule difficult to assess when the patient

is giving us an honest expression of his or her symptoms even though there may at times appear to be considerable discrepancies. In no condition is it so important that direct contact with the patient be established and where the actual description of the intensity or anxiety experienced be only conveyed directly by the patient. Levy,(11) in his book stresses this point that a "second-hand description" of the symptoms is often of little value and that no reading of notes or records can quite take the place of hearing the patient's own story.

Dr. John Parkinson in discussing this type of case states that it is not "where the pain is, but when it is" that matters, and this appears to be the outstanding feature in the diagnosis of anginal pain, although certain definite and suggestive facts can be obtained from the consideration of the character of the pain.

I have found in this condition an almost unlimited variety of descriptions of the pain distribution and causation, and herein lies the intense difficulty facing us in the consideration of this particular malady.

CHARACTER OF PAIN.

Usually the patient comes to the consulting room complaining of a pain varying in intensity from a mere discomfort which hardly makes much difference to the

normal routine at one end of the scale. At the other end of the scale the patient will describe a gnawing, twisting, throbbing, vice-like pain, which causes him to stop for a few minutes or longer according to the intensity of the pain.

The description of being "gripped in a vice" or the sensation of the chest "being pressed upon" with such considerable force that even breathing becomes difficult is a frequent finding. Occasionally the pain is described as "twisting" in nature by some patients in their attempt to put into words what must indeed be a pain of frightening intensity. Of all the variety of descriptions of the pain, one feature seems to stand out, namely the continuity or continuousness of the pain.

As we shall see later many cardiac and non-cardiac pains can produce discomfort of a similar nature, but usually we find some alternation in the pain, and some intermitteney or "stabbing" characteristics. In coronary insufficiency, once the pain has been estblished it continues without interruption although with gradually diminishing intensity on rest or on the application of treatment.

When we consider this feature in the light of Lewis' experiments on "Pain production in Ischaemia Muscle" the great similarity between the two types of pain becomes

very obvious. It has always been a matter of some surprise how patients will continue with this condition often for many months or longer before medical advice is sought. In most instances the explanation usually is that the patient attributes these pains to "indigestion", and in fact, it is not unusual to find him prepared to associate many discomforts of this type with gastro intestinal causes, and that somehow the stomach seems to be the "root of all evils". There is however some justification on the patient's part for this assumption, since we do know that attacks of anginal pain are often associated with gastric symptoms. We know also that anginal attacks are more likely to occur in association with a meal since we have as the result of the latter a reflex reduction in the coronary flow. We must also bear in mind the possibility of displacement and rotation of the heart following a full meal with consequent impedance of the coronary circulation.

Many of these attacks are associated with eructation or with subsequent aerophagy and it is therefore very natural on the patient's part to feel that he is suffering from indigestion and in many instances this accounts no doubt for the patient's attempt to "work off" the attack. It is often surprising to find how many patients will give a history of being able to carry on in spite of the pain although having to reduce the rate of their efforts. This we

will admit only occurs in a few early cases where we might describe the condition as a discomfort rather than a pain. The big majority however, do find that the condition will not disperse by further exertion although we find cases where very drastic results in the form of obvious myocardial infarction have followed in the case of a determined patient who will not give in to the pain.

The term "angor animi" or fear of death used to be associated with the condition but it is now recognised as a very occasional accompaniment and indeed is not uncommon in other conditions. However, there is no doubt that the sense of constriction of the chest does appear to give a "fearful" aspect to the pain and often intense mental anxiety is experienced, occasionally bringing the patient to tears. The inability to talk or speak to anyone whilst the attack is on, is a characteristic feature of this condition although it is only found in advanced cases.

DISTRIBUTION OF PAIN.

In trying to assess the distribution of this pain we can place the various localities in order of frequency encountered in practice, and here we find ourselves faced with a much greater variety than in our discussion on the character of the pain. Nevertheless in spite of this, certain well defined areas of distribution can be ascertained

which are of a great help in the diagnosis. We find a tendency on the part of certain literature to use the term precordial in discussing cardiac pains generally but this is a very misleading term to use at any time. The word precordial means covering an area over the heart and has a disadvantage of implying a wide area which might be either front or back, since we have a precordial area on the posterior aspect of the chest wall. In fact some authorities actually suggest that the auricle can be percussed in the back, although there is some doubt if this can be reliably performed. The words supra mammary infra, or submammary, substernal and so on are much more suitably employed in an attempt to define exactly pains of this nature, or if vague, the word cardiac pain is more suitable.

Usually we find that the patient comes to see us complaining of this characteristic pain in some area of the chest following exertion or emotion, and it is extremely useful to ask the patient to define the area by pointing to the seat of the pain with one finger and it is surprising how often this procedure can clarify an otherwise vague description. The classical description of pain of coronary insufficiency usually implies that the pain is substernal in type.

1. SUBSTERNAL PAIN.

This we would be inclined to rate first in order of precedence and usually involves a pain either radiating down the whole length of the sternum or located in the lower end of the sternum or just over the xiphi-sternum. At the same time we are by no means prepared to accept this as a frequent occurrence, and this type of pain is followed a close second by pain of the supramammary type.

1. SUPRA MAMMARY TYPE.

Here the patient will point to an area a little to the left of the sternum and somewhere about the third or fourth rib, sometimes shewing radiation into the left axilla. This type of pain can often be quite localised, with radiation to the left mid axilliary line or can be of a more diffuse nature covering more or less the areas of the second, third or fourth thoracic nerve roots. Both these types of pain are found in association with certain well defined characteristic radiations, and next in order of frequency we would be inclined to include distribution either substernal or supramammary with one or more of the following radiations, although more often than not there is no radiation. (Lewis)

2. Radiation into Arms.

The usual radiation of anginal pain noted in this series is on the inner aspect of the left arm as far as the left elbow only, and we have been surprised to find how many cases will give this particular type of distribution coupled with 1 or 2.

3. Radiation extending throughout arm to fingers.

This type of distribution we are inclined to classify next in order involving usually the left arm and following the distribution of nerve C7 and 8 as far as the fourth or fifth finger of the left hand. This is a fairly common distribution.

4. Radiation into both arms.

This is a comparatively infrequent distribution but does occur and when it does it usually involves the ulnar distribution in both arms.

5. Radiation into the back.

Next in order of frequency we could specify as radiation into the posterior aspect of the left chest wall usually subscapular. This type of case can be on occasions most misleading, and we must bear in mind that it is not uncommon to find a pain of this type with radiation

into the posterior axilliary region described as the prime focus of pain.

6. Radiation into posterior aspect of arm.

It is suggested by some authorities that the posterior aspect of the arm is seldom involved but we could not endorse this from our experience. Quite a few cases were found where the patient described the distribution of the pain as extending from the left shoulder down the outside of the arm to the elbow - in fact we might say this distribution is really surprisingly common.

ABERRANT FORMS OF DISTRIBUTION.

Next we come to a series of unusual distributions of pain which are perhaps the most difficult of all to elucidate. First of all we might classify these as follows:-

7. Radiation into neck.

This particular form of unusual distribution usually results in pain in one or both sides of the neck in the posterior aspect (usually both) and can sometimes produce muscular spasm of the Scelenus Anticus muscle and can simulate spasm of this muscle due to other causes. One case was recorded where pain was experienced on the left side of neck for 14 days prior to the occurrence of a

myocardial infarction, and here the pain only became substernal two days before the acute incident.

8. Radiation into the Jaw.

This is not common, but can be misleading when it does appear, and can often be the only form of pain occurring on exertion.

9. Epigastric distribution.

This is an occasional aberrant form which can often be confused with gastro-intestinal conditions although it is usually associated with a certain amount of pain over the lower sternal region.

10. Abdominal pain only.

This group can be exceedingly difficult when encountered, and only recently a patient was seen complaining of pain in the right hypochondrium following exertion and relieved by Nitrites, with definite Electrocardiographic changes (BBB) and hypertension. This can be simulated in some cases by patients shewing some degree of failure with slight enlargement of the liver.

BILATERAL DISTRIBUTION OF CHEST PAIN.

We must bear in mind however the patient who complains of a pain stretching across the whole chest during an attack, usually however if we adopt the policy

of asking the patient to point with one finger, we usually find that the predominant focus of this pain is either directly substernal or left supra-mammary, but the presence of bilateral pain, is more suggestive of thrombosis.

LEFT INFRA-MAMMARY PAIN.

We have purposely left this particular location of pain to the last of this series, since of all the pain foci it is least frequently associated with pain of coronary disease. In fact the presence of the pain in this location is always much more suggestive of a pain of non-anginal type, but again not invariably so.

HYPERAESTHESIA.

The presence or absence of Hyperaesthesia of Hyperalgesia can be useful in differentiating anginal pain from other conditions. Some deep tenderness does persist for a very short time following an anginal seizure but is usually transient and its continued duration makes us suspect the occurrence of coronary thrombosis. The persisting ache for hours or days is more characteristic of cardiac neurosis although rare variations can occur. Numbness in the arm does exist following attacks of anginal pain and one patient was seen who described

severe mottling of the skin both after and prior to attacks of anginal pain that day. It is suggested by some authorities that the pain which only amounts to a slight discomfort is just as serious from the point of view of prognosis as the pain of considerable intensity. This raises this very vast question of the extreme variation in sensitivity on the part of different individuals to pain impulses. We know that such great variation and sensitivity does exist in certain individuals in a quite inexplicable way and is by no means a matter over which the patient has any degree of control. Recently a case of Haematemesis was noted in a patient who we might have felt inclined to describe as a hypersensitive type. On X ray investigation an extensive duodenal ulcer was found although the patient had been completely devoid of any gastric symptoms. This type of case is commonly seen in general practice and is illustrative of this peculiar variation in sensitivity of different individuals to pain.

Millar[13] discusses this question of the sensitivity of the individual very fully in his book. He draws our attention to examples of primitive rights exorcised by various South American tribes where environment and upbringing is such that a considerable degree of stoicism has been evolved. Heredity and disposition do weigh considerably in this particular matter since we have

wide variations in the stability of the nervous system in different individuals. At the same time we feel more inclined to think that it is a fortuitous occurrence that certain people do have a higher threshold for pain than others. Of course we know that certain people are inclined to make more fuss over pain than others. We might in this discussion raise the whole question of the difference in sensitivity of the two sexes, and it seems a well recognised fact amongst cardiologists that cases of anginal pain are brought to notice more readily in the man than in the woman. The capacity to tolerate pain is not so great in men, probably because he is of a more complaining nature and is less tolerant of any discomfort.

THE OCCURRENCE OF THE PAIN.

Generally speaking this pain can be induced by effort or exertion or some particular emotional upset. These are the usual accepted causes, but a very wide variation exists in the degree of effort required in different patients. On exertion we naturally have as the result of increased oxygen demand on the part of the heart a greater disproportion of supply and demand of oxygen via the coronary circulation. We must regard this particular

feature of "when" the pain occurs, as one of the corner-stones in our diagnosis of coronary insufficiency. Some authorities make an attempt to differentiate between various degrees of effort inducing the pain.

Vasquez (14) is quoted by Miller as supporting the division of anginal pain into two groups viz:- angina of effort and angina of repose. Daniel Opu in this same article is quoted as further subdividing angina decubitus into angina of repose while awake and angina of repose while asleep. This seems to me to be an unnecessary sub-division since all are manifestations of one and the same condition in various stages in its progression. At the same time we must admit that pure cases of Angina Decubitus can occur in bedridden cases of advanced aortic disease. We do know also that typical attacks of anginal pain do occur during sleep as the result of some intense emotional storm with its consequent effect on coronary circulation, and where these cases demonstrate "Angina of Effort" while awake.

The pain is usually induced by effort or emotion of an exceedingly varying degree. One patient was seen who could induce an attack while picking up her handbag from the floor and yet could manage, with quiet walking for a reasonable distance to avoid the onset of pain. This is an

unusual manifestation and as a rule pain induced by arm movements only is rather suggestive of an extra cardiac cause. At the other end of the scale cases occurred where only lifting heavy weights or doing heavy work such as gardening or sawing wood could precipitate an attack. In between these two extremes we found that the majority of cases induced pain by walking, climbing or hurrying etc. One feature of this condition which has always been a certain amount of surprise is how we can get such extremes of effort producing pain in the one patient as quoted above.

Miller[15] suggests that great variation of exercise tolerance can occur in each individual, but we would be inclined to differ with him on this point. There are certainly many cases which apparently suggest that this might be so but on a more careful enquiry a very considerable degree of constancy of the pain for each individual can be found. Many patients will certainly tell us that they may go for some days without pain but on careful enquiry we will usually obtain the admission that they have restricted their exertion and perhaps have not been doing the type of exertion which normally brings the pain on. We find that if the patient has some particular exertion which he must do daily. a history of constancy can usually be obtained. The type of pain induced say on climbing the stairs to the station first thing in the

morning or hurrying to catch the train is often a very useful guide in diagnosis. This type of pain induced on exertion in the morning soon after a hurried meal and constant in its occurrence can be a very useful indication here. Usually the pain appears later in the day on some similar exertion and there is no doubt that the relation of the exertion to the early meal particularly at a time when the atmospheric temperature is lower produces a combination of circumstances most likely to show up any diminution in the coronary supply. A similar situation often arises at night with a fall in temperature and one patient was seen who invariably had an attack of pain on going out for a walk in the evening.

At the same time we must be prepared to admit that wide remissions in the intensity of the condition and in the degree of exercise tolerance do occur, and in fact we might almost say that a seasonal variation can be observed in some cases probably due to atmospheric temperature and pressure.

Mackenzie discussed this factor of variability and admitted that it was impossible to account for it. He found that the attacks disappeared for week or months at a time but he felt that sleeplessness, worry or systematic over exertion could induce a recurrence of the attacks. Nevertheless, we are always more inclined to suspect cardiac neurosis as against anginal pain where

frequent variation in onset and intensity occur.

We have already hinted that cold is a factor in the diminution of exercise tolerance and it is surprising to find how frequently this well recognised feature does occur in a series of cases investigated. Ths history of pain on going upstairs into a cold bedroom or entering a cold bed or getting out of bed during the night is amazingly common although we have often got to put a leading question in order to ascertain this fact.

We do know from various experiments that T wave changes can be produced in the electrocardiagram by drinking ice cold water and ST changes also have been observed. Another factor influencing the frequency of attacks is the relationship of effort to meals. It is a well recognised fact that even the normal healthy subject can experience some substernal discomfort following violent exertion after a large meal. We know that the demand on the part of the splanchnic vessels can reduce the coronary circulation and therefore it seems obvious that where some impairment of the coronary circulation is already present any reduction in the coronary flow will make itself felt at this time. At the same time, the liability to confusion of substernal pain with indigestion makes this a very disconcerting feature which will be discussed later.

We are therefore prepared to admit that a certain variation can occur in the facility with which these pains

can be induced, but not of sufficient magnitude to disturb what we must regard as one of the dominant features of the diagnosis of anginal pain namely the relative constancy for each individual of the amount of exercise or emotion required to produce the pain. Emotional upset was not found to be a very frequent cause of this type of pain and where it occurred as a sole cause was extremely rare, but it was not uncommon to find such patients have an attack during the air raids experienced here. It is suggested by some authorities that where emotion is the prime cause of the pain the condition is a comparatively benign one, and support was found for this statement.

The next important point in considering pain of this type is the relief of pain with rest, and this seems to be a fairly reliable and constant feature. The time varied from a few minutes to 30 minutes or more and some even longer, where no actual coronary thrombosis appeared to have ensued. This of course is a very difficult matter to discuss, since it is obvious that where the ischaemia is too long continued thrombosis or even minute areas of thrombosis may be occurring from time to time. It seems feasible to assume that where the duration of the pain is longer the myocardial ischaemia must obviously be of greater extent, or perhaps the flexibility of the coronary system may be more impaired. Some authorities suggest that a duration of over 15 minutes must make one suspect that

coronary thrombosis has occurred, but this is much too stringent a test, although it would appear that a pain of short duration, other things being equal, should be regarded favourably.

The whole theory of "second wind" in relation to the the pain of 'angina' raises a point that is worth further study. We know that in the athlete the occurrence of "second wind" is a physiological reaction to extreme exertion, and it has been suggested that this is due to some dilatation on the part of the coronary vessels. The patient will have an attack on first going out to business in the morning or will have to pause during his first walk of the day and then will be able to continue with greater effort and less liability to pain. The tendency to stop and look in a shop window or to pretend to "look at an aeroplane" till the attack passes off is of course a regular feature of this condition.

It has been suggested by some that where one comes across this "second wind" history that it means a greater coronary reserve, and that the coronary arteries are not sufficiently sclerotic to prevent their dilatation but the possibility of this being indicative of a good prognosis seems to be doubted by most. Although the mechanism here seems to be somewhat obscure, various writers on the other side of the Atlantic seem to lay quite considerable stress on the favourable prognosis

of such cases.

The important feature from the diagnostic point of view in the consideration of the pain in coronary insufficiency is what relief is obtained from the pain and sense of constriction either immediately on rest or soon afterwards.

VASOMOTOR SENSITIVITY.

Mackenzie states that "some people have a nervous system unduly sensitive to stimulation". Many cases give a definite history associated with vasomotor instability with a variety of changes sometimes prior to, during or after the attack. It is very natural to assume extreme sensitivity of this type, and in fact many of these cases where no other organic cause can be found, must be regarded as being due to an intense vasomotor spasm. This we know to be the case by record of the blood pressure readings, taken during attacks, and where in some cases vasomotor crises of 320mm systolic have been found. In fact the extreme rapid rise of blood pressure during an attack and the rapid fall afterwards imply vasomotor sensitivity as a background to this condition and this blood pressure change is one of the diagnostic features in contra distinction to the persisting drop in pressure in coronary

occlusion. We find vasomotor changes such as migraine, hemianopias, hemicranias, flushing, tingling, etc. during and following an attack.

HYPERTENSION.

The association of hypertension with the anginal syndrome is a very important one and where it is found it is a useful guide in diagnosis. Lewis maintains that in the first instance the disease is part of an atheromatous change in the vessels of the body generally and in the coronary arteries in particular, and Levy maintains that arteriosclerosis is the most frequent cause of coronary thrombosis. An investigation by Parkinson & Bedford into one hundred cases of anginal pain (private & Hospital) found forty nine per cent to have hypertension. Hypertension can exist apart from arteriosclerosis but it seems to be generally accepted that continued hypertension does produce arteriosclerotic changes in the blood vessels. Levy shews that arteriosclerosis can occur at any age and that diet, infections, tobacco and indeed many toxic factors can be found to be responsible. The association of arteriosclerosis with diabetes, glomerulo nephritis and hyperthyroidism is easily demonstrable. There seems to be no definite proof that worry as a cause can be directly attributed to the production of hypertension, but heredity does appear

to play a very considerable part indeed. This subject is discussed more fully on considering coronary sclerosis.

DYSPNOEA.

Most cases with coronary insufficiency and anginal pain complain of dyspnoea as a dominant sympton and it is usually suggested that dyspnoea as an indication of failure terminates the pain, this is not altogether true in practice. It is by no means uncommon to find both dyspnoea and anginal pain exist together but as a rule the combination is more frequent in the ageing patient with some indication of either valvular disease or myocardial disease or both.

Response to Nitrites.

The response to coronary vaso-dilators such as trinitrin, amylnitrite, or cardophyllin can be useful as a guide to diagnosis in doubtful cases, although frequently the response is not always absolute.

Some cases state that relief is definitely obtained while others will describe a great diminution in the pain but some residual discomfort. Where relief is not obtained we are always inclined to doubt the diagnosis of coronary insufficiency. We usually advise the patient to chew the tablet frequently and if possible before undertaking any exertion which normally causes the pain. The

experienced patient usually does this of his own accord and often will be found to chew a tablet say in the cinema prior to his walk home. In the reasonably intelligent patient the response to trinitrin can often be of decisive value in diagnosis, and cases even get relief from a tablet taken night and morning only. In view of the comparatively transient action of trinitrin (two hours at the most) this has always been a matter of considerable surprise to me. In the definite case of anginal pain due to coronary insufficiency relief is usually obtained within a few minutes unless coronary occlusion has complicated the picture, when of course no response is obtained from this remedy.

DURATION OF SYMPTOMS.

A great variation in the duration of the symptoms is commonly found in this condition, although I find a peculiar constancy in the period of time elapsing before the patient comes to the consulting room. Often three to six months can elapse before a patient decides that the recurring pain cannot just be a form of indigestion, although of course a fair number are seen within days or weeks of onset, particularly if that onset has been heralded by a coronary thrombosis.

It is surprising however to find certain cases continue for many years with little or no aggravation of symptoms and I know of one case where definite anginal symptoms have been experienced for at least 26 years with no apparent increase in intensity of frequency. This particular patient is an example of how careful regulation of work can prevent deterioration in such conditions. He was seen at the Heart Hospital many years ago and advised to reorganise his life within the range of his coronary capacity and now never does anything liable to induce his pain. This type of case is no doubt a great exception and as a rule some tendency to deterioration can usually be found. The whole guide to prognosis in these cases is undoubtedly the rate at which exercise tolerance is increasing or decreasing. It is often useful to try to assess the distance in feet or yards which the patient can walk before inducing the pain, and some comparison can usually be obtained in this way. Where the pain is only produced by say walking uphill against a wind it seems that the outlook is better particularly if the resulting pain only amounts to a substernal discomfort. On the other hand where exercise tolerance is waning rapidly and such slight movements as coughing, sneezing etc. induce the pain, then the outlook is unfavourable, and sooner or later "angina decubitus" with terminal occlusion may ensue.

Many cases of anginal pain however do improve and in some cases the symptoms have been known to disappear completely. This is more usual in angina of effort following a first coronary occlusion where perhaps recanalisation of the clot or establishment of collateral circulation ensues.

Rare cases also have been known where "angina of effort" has completely disappeared following a coronary occlusion with, it is assumed, destruction of the nerve endings in the area of muscle previously the seat of ischaemia.

DIFFERENTIAL DIAGNOSIS.

In the investigation of cardiac pain in general practice we must firstly consider its differentiation from other types of thoracic pain which can so often closely simulate it. In practice this can present one of the biggest problems in medicine, particularly when such cases are seen in the routine of a busy practice and where no special facilities are available for detailed investigation.

We can roughly divide other similar pains into two main groups namely (1) Those of cardiac origin and (2) Those due to extra cardiac causes.

CARDIAC CAUSES.

Myocardial Infarction.

Of these perhaps the condition which is most frequently encountered and confused with anginal pain of chronic coronary insufficiency is myocardial infarct. In fact we are quite safe in saying that in a certain proportion of cases actual separation of the two is impossible, since both conditions are due to the same cause.

It is indeed a fact that a very large percentage of cases of chronic coronary insufficiency are associated with infarction due to occlusion at some time in their history. It has even been suggested by some authorities across the Atlantic that a history of myocardial infarction would be obtained in all cases of angina of effort but we have every reason to doubt such a sweeping statement, since cases are frequently encountered where no indications either clinically or by electrocardiograph investigation can support the occurrence of an infarct.

Usually the history is quite different from that of chronic coronary insufficiency and an abrupt onset and pain of much greater persistence is more frequent than in the more benign condition but nevertheless great variations can be found. The pain usually persists for an hour or more and even for days and a considerable degree of sub-sternal ache ensues. This precordial hyperalgesia is

a very useful indication and is not found to the same extent in chronic coronary insufficiency, and we could go so far as to say that in a known case of angina the occurrence of intense hyperalgesia following a paroxsym rather implies that coronary occlusion may have complicated the picture. Hyperalgesia of course, is a frequent accompaniment of the left submammary pain of the non-anginal type, but is an unusual accompaniment of the true angina of effort.

The pain of infarction often occurs suddenly during the night or following some severe exertion and is usually associated with a high degree of collapse or peripheral shock and vomiting. At the same time it is customary for the patient to attribute his attack to "acute indigestion" and not infrequently the patient has already worked out to his own satisfaction the particular food stuff which has caused the attack. The pain is often entirely substernal or epigastric but a pain across the chest or even confined to the left supra mammary region with radiation into one or both arms is not uncommon.

The restlessness of the patient is a characteristic feature of the condition distinguishing it from the motionless attitude of angina. No relief from nitrites is obtained and usually some morphine preparation is required. Signs of a temporary cardiac failure such as some oedema of the lungs with palpable liver or spleen can be found. A low blood pressure is almost invariably an accompaniment

although this may not occur at once, but when it does so it is usually sufficiently great to make a sharp distinction from the considerable rise found during the anginal paroxysm. Changes of rhythm and rate are often found and although usually of a temporary nature make the prognosis less favourable. A rise in temperature can often be ascertained after about 24 hours, but this can sometimes be very slight.

Leucocytosis of 10000 or over is a useful indication and it is suggested that anything about 20000 indicates a massive infarction with poor prognosis. The rapid rise of sedimentation rate and slow fall are supposed to indicate a good prognosis, but occurs in other conditions.

A pericardial friction rub after about 24 hours may occasionally be heard, but is rare, and only persists for a day or two at the most, but it can make the differential diagnosis from acute pericarditis very difficult. The latter however is usually more widespread and does not disappear in such a short time, although the pain and similarity of ST changes in the cardiograph can be very confusing. A silent thrombosis can occur with similar rise in temperature and associated symptoms, but with no pain; and it is here that the electrocardiograph can be of the utmost value. Some cases can be sufficiently benign or the patient's threshold for pain sufficiently high for a coronary thrombosis to pass almost unnoticed or at least

unheeded. Only a day or two ago a patient appeared at the Heart Hospital with a story of momentary collapse while performing an acrobatic feat on the stage in a London theatre. This patient then continued with the performance but he presented himself at the Heart Hospital two days after the occurrence where an extensive myocardial infarction was diagnosed, and he was surprised to be told that he had to return to bed at once.

Many patients unfortunately do not seek medical aid at all and carry on often until angina of effort ensues about a month afterwards, and a history of persistent pain at the onset can often be obtained only after a series of leading questions.

It is suggested that at least fifty percent of cases of "angina of effort" can be found to shew evidence of old myocardial infarcts on electrocardiograph investigation, using the classical chest leads and there seems little doubt that using the posterior chest leads a much higher percentage will obtain. Coronary thrombosis is most frequently found as a precursor of angina of effort and some degree of angina usually follows all cases of thrombosis, but often diminishing after a few months. Conversely a fair number of cases with a history of chronic coronary insufficiency develop a coronary occlusion at later stages and often as a terminal condition but it is more or less impossible to tell which cases are going to develop thrombosis. However, age and

the existence of hypertension are found to be definite predisposing factors in the production of occlusion and cases over fifty years of age with high systolic or diastolic pressures shew a high incidence of coronary occlusion. This type of case illustrated below is not uncommon:-

Mr.T. aged 58 years. A Commissionaire. Seen during an attack of severe and constant pain over the left breast radiating to ulnar aspect of left arm and of 24 hours duration. This pain was induced by an attempt to saw wood in his garden. Patient was obviously in great distress and respirations were rapid and laboured. Cough with rusty sputum was also observed. Previous history of substernal pain on exertion such as lifting, or fast walking not lasting longer than 20 minutes. This condition had obtained for nearly 10 years and he had been most careful to avoid onset of pain, and had always inhaled amylnitrite during paroxysm which gave him relief.

Examination. B.P. 140/90 but definite history of hypertension 185/? obtained.

Heart sounds faint with definite signs of consolidation of left base.

Rate 72 Regular. Temperature 101

Clinical diagnosis was made of coronary thrombosis with left basal pneumonia.

ECG showed typical changes of posterior basal infarct.

He was finally admitted to hospital when I understand a second thrombosis occurred which proved fatal.

In those cases where coronary thrombosis has ensued the patient is usually conscious of a difference in the intensity and character of the pain from his usual anginal paroxysms. In the case just quoted the patient remarked that he always had palpitation after his attack of anginal pain but no such occurrence had been observed on this occasion.

It seems very obvious that each attack of myocardial ischaemia no matter how transient it may be might induce some degree of myocardial damage and that repeated attacks can gradually produce a myofibrosis which sooner or later will lead to failure. Herein lies the great importance in advising the patient to avoid the onset of pain at all costs and the advantage of a reasonable period of rest following an attack. Herrman (22) advises the patient to rest as long as ten times the length of the pain and points out that those cases do best where a readjustment of their lives to a lower tempo is possible. The differentiation of two closely related conditions namely the anginal pain of chronic coronary insufficiency and the mild coronary occlusion can be extremely difficult. The distribution of the pain is relatively similar although much more persistent in the coronary occlusion and often more epigastric in type. The spread of the pain of an acute coronary occlusion can be most confusing when the abdominal form of radiation is experienced.

The possibility of an acute cor pulmonale occurring and simulating closely coronary thrombosis must be borne in mind although here some focus of emboli such as the leg or some post-operative state can usually be observed.

We can roughly summarise the characteristics of coronary occlusion which can help in differentiating this condition from coronary insufficiency alone.

(1) It is usually more intense
(2) Much greater persistence
(3) The shock is greater
(4) Occurs during night or after severe exertion
(5) Associated signs of temporary failure
(6) Disordered rhythm frequent
(7) Restlessness of patient
(8) Rise in temperature
(9) Fall in blood pressure
(10) Leucocytosis usual
(11) No response to nitrites
(12) Rising sedimentation rate
(13) Electrocardiograph findings usually positive

CARDIAC NEUROSIS.

Perhaps the next condition in order of frequency which is liable to be confused with the anginal syndrome is cardiac neurosis in one of its many varied forms. Some of these conditions at one time were labelled "pseudo angina" but this term is a definitely misleading one and

the term angina should never be employed because of its association with a serious and incurable malady in the mind of the lay public. In the above group of cardiac neuroses with pain we have various vasomotor spasms, the so called effort syndrome, and neuro vascular asthenia and possibly so called nicotine angina.

Here we have a pain which although in many ways like that of true angina is usually more diffuse in its distribution and much more varied in its character. Extreme inconstancy of occurrence of the pain is a dominant feature and as a general rule the pain is left sided and usually confined to the left submammary area. Although in angina we do get considerable variation in the day to day incidence, in no way is this variation comparable to that of cardiac neurosis. Bramwell[23] likens the irregularity of the symptoms in neurosis to that of nervous dyspepsia in which the patient is extremely ill one day and comparatively well the next. He stresses here the fact that although the patient with angina may transgress his normal limitations with impunity when his mind is preoccupied just as the athlete may surpass his performance in practice under the emotional stress of the contest, yet broadly speaking the exercise tolerance of a patient with angina remains remarkably constant.

This however is not the case in the cardiac neurosis and this great variability is indeed most helpful in discriminating between these conditions. Radiation of the pain in neurosis is not very frequent but does occur. The pain itself is usually described as a sharp stabbing pricking sensation or sometimes as a dull ache in the left submammary region, but radiation to the back and in some cases to the left arm is found. Bramwell stresses the symmetrical distribution of the pain of angina as against the asymmetrical distribution in neurosis, but this is not a reliable guide, since many cases of anginal pain are found confined to left side only. Lewis (25) suggests that in enquiring as to the character of the pain it is useful to leave a decision until the patient has had occasion to pay special heed to the particular points raised and this is a very useful suggestion since many patients can on first questioning be surprisingly vague about their symptoms. Lewis is inclined to stress more the purely substernal character of the anginal pain and does not emphasise unduly its symmetrical character. We must bear in mind the possibility of a cardiac neurosis superimposed upon definite organic disease and this is by no means infrequent. Another big problem is the ease of access to medical literature which a patient (particularly of the neurasthenic type) can obtain to-day, and which definitely encourages him to "gild the lily". Such a case as the following illustrates this fairly well:-

Mr.JCB. Aged 35, A factory worker, complained of three attacks of pain in his chest since 1941, the last attack 3 days ago. Pains described as coming on suddenly and of a "shooting" character lasting 2-3 minutes and passing off gradually; followed by "twinges" of pain for a few hours afterwards. Sometimes described pain as "bursting" in nature.

He has had periodic blackouts. During attacks he described "waves of pain" passing down the left arm sometimes to the elbow and again involving the whole hand which goes numb. Pain came on while sitting quietly in chair and seemed to have no relation to exertion.

B.P. 125/60. Rate 65.

Tendency to obesity and sweating.
Knee jerks exaggerated.
There is a considerable neurasthenic background in this case.

ECG findings were negative, except for left axis deviation.

This type of case is, in all probability, a cardiac neurosis with a superimposed exaggeration of symptoms, the latter, no doubt, being intentional.

We discharged him from hospital and told him that he had no heart disease and he has now returned to work; and when seen two days ago was not complaining of any discomfort.

Cardiac neurosis is a more frequent occurrence in the young woman and it seems that true anginal pain should be diagnosed very rarely in the woman at all, before middle age. One feature which was observed frequently was the occurrence of pain after the effort especially when the patient was tired and often persisting for a

considerable time. In the anginal pain the onset is usually during the effort or immediately after, and is relieved soon by rest. The severity in neurosis is never nearly as great as in the anginal type nor is the peculiar sense of constriction of the chest so intense. In these cases one usually found an associated history of overwork or excessive worry associated with various vasomotor disorders. Flatulence or heart-burn, palpitation and shortness of breath with sighing respirations often appear in conjunction with the pain of neurosis. It was customary to find the patient complain of inability to take a deep breath and hyperalgesia of a wide precordial area was exceedingly common and was a useful diagnostic feature in the comparison with the anginal syndrome proper, although exceptions to this rule can always be found. As an example of this type of case we have the following:-

> Mrs W. aged 42 years. Complained of periodic attacks of aching pain under the left breast radiating into the left axilla and sometimes also to the posterior aspect on the left side. Occasionally radiation was experienced to the left shoulder and posterior aspect of arm to the elbow. Had frequent numbness of hands and feet and often pronounced sweating of extremities. Was short of breath on climbing stairs but only gets pain if tired, usually after sitting down. This patient dated her whole condition from the loss of her only son at Dunkirk.

Examination. Sighing respiration was noticeable.

B.P. 150/90 rate 80 regular.

On Auscultation accentuation 2nd aortic only finding.

ECG normal.

This patient stated that her condition was always aggravated at period times and this was a common complaint in this condition. She had also a history of profuse bleeding from cuts or dental extractions and at period times. It seemed that this might have some connection with her other vasomotor manifestations.

As a rule we find a neurotic woman with an unstable nervous system either as the result of physical or mental fatigue or a hectic existence with a tendency to palpitation or tachycardia. Cases of this type were frequently seen during the period of the heavy bombing of London as the result no doubt of the constant stress at that time. We must be careful not to confuse this type of case with the tachycardia of hyperthyroidism since tachycardia as a rule is one of the prominent features of the cardiac neurosis.

EFFORT SYNDROME.

This condition is a form of cardiac neurosis originally regarded as essentially found in young men thrown into the stress of war who had previously led very

sedentary lives. We must remember however that effort syndrome is by no means purely confined to soldiers and many cases of it are found in civil life. A left sub mammary pain with palpitation, breathlessness and giddiness are perhaps the outstanding feature. It seems that the differentiation between effort syndrome and neuro-circulatory asthenia is very difficult and very often both conditions co-exist. The exaggerated response to effort and pronounced dyspnoea on moderate exertion without any underlying organic cause, are frequent manifestations with or without left submammary pain. Hyperaesthesia or hyperalgesia of a persisting type is a common accompaniment also. Frequently some definite toxic factor can be located and the incidence of incipient tuberculosis has been found to be high in this type of case. As a rule a considerable nervous instability provides the background and the exaggerated response to effort can often be extremely helpful.

Lewis[26] suggests a simple hopping test which raises the normal heart rate of healthy young people to 90 or 100 subsiding in a minute or less. In milder cases of effort syndrome he states that a jump to 120 or 130 or even much more is to be expected, with a subsequent delay in return much beyond the normal. This is an extremely complex condition and to quote Dr.John Parkinson "the mere fact that we can call it "effort syndrome" does not imply that

we know any more about it." Cases of "effort syndrome" associated with the stress and strain of war are quite common to-day.

The following case makes an interesting example:-

Mr.A.P. Aged 23. Rate 96 normal rhythm. First complained of irregularity of heart while resting or running in 1940. At this time was stationed at Reykjavik, Iceland, where he was admitted to hospital for observation but finally discharged after negative findings.
He is a very sensitive type of youth and was greatly upset by his experiences as a soldier in Norway where he had to march on one occasion 59 miles in 36 hours. Now describes a stabbing pain under the left breast with frequent bouts of palpitation and complains of fatigue. On examination no abnormal findings were obtained.

ECG showed a few auricular extrasystoles with tachycardia.

This case is interesting in view of a recent letter in British Medical Journal on the frequent occurrence of effort syndrome in Iceland. The possibility of the long winter nights with their opportunity for introspection and the arduous nature of the country are discussed as a contributory factor but not necessarily the cause.

The exaggerated response to effort which is more frequently found in neuro-circulatory asthenia might be illustrated by the following case:-

Mr.M. Aged 18. Aircraft worker. Rate 72. Seen complaining of pain under left breast with occasional "giddy turns"; got out of breath easily if he hurried and complained of being easily tired. States also that on deep breathing a pain was experienced in left submammary region. Fine tremor of hands and knee jerks exaggerated.

Auscultation apical systolic murmur with double or split first sound.

Pulse rate on resting 68 but on hopping test 120 returning to normal in over two minutes.

B.P. sitting 130 systolic Diastolic 60 but on standing 110/50.

Some degree of orthostatic hypotension exists here.

Some such cases have been known to shew an inversion of T3 or even T3 and T2 but never T1, although this is a rare finding and as a rule in these cases little abnormal findings are obtained except perhaps an occasional systolic murmur with a diffuse impulse at the apex. Usually some degree of coldness of the hands and feet or free sweating of the axillae or some other vasomotor abnormality is present.

Herrman (27) states that it is a common occurrence for a patient to admit that he has never paid much attention to his cardiac condition until he was informed by some examining physician, of the existence of a cardiac

disorder. This no doubt, is only too frequent and we must bear in mind that the existence of a genuine cardiac lesion must facilitate the development of a cardiac neurosis and it is not infrequent to come across both conditions in the one patient. Hermann quotes Viko & Conners who have made a study of this particular field and who are convinced that suggestion from the physician was invariably the precipitating factor in the development of a cardiac neurosis where a neurotic background exists.

Lewis (26) stresses the fact that many of these cases of cardiac neurosis are actually suffering from unrecognised chronic infections and that the condition of their nervous system is such that any pain is poorly tolerated. In this same article Lewis stresses the much greater frequency of cardiac neurosis amongst women under 45 years of age and insists that a diagnosis of angina should be made very infrequently in this class of patient.

The co-existence of a cardiac neurosis with a cardiac lesion might be illustrated by the following case:-

Mrs S. aged 47 years. Had history of recurrent attacks of rheumatic fever since 17 years of age. Now complained of left submammary pain with radiation down inner aspect of left arm to the elbow. Sometimes all the fingers of the left hand would go dead. Pain could be induced by exercise such as lifting or such emotional upset as a fright. It often persisted for hours leaving a definite tenderness over the whole precordial area. Frequently the pain occurred at night or on resting when she was tired and was accentuated at period times. Attended "Womens Hospital" for similar condition in 1918. Was quite definite that no fixed amount or type of exertion can induce the pain and it often disappeared for months only to return if she was excessively worried.

Auscultation Presystolic murmur at apex with accentuation of first sound.

B. P. 105/85

ECG normal

Here is a case of a mitral stenosis with what appeared to be a superimposed cardiac neurosis although some of these cases can be extremely difficult to assess.

Dr. John Parkinson in discussing the follow up conducted on the majority of cases of so called D.A.H. in the last War states that the big majority were still found to be in good health 20 years afterwards, although many were still complaining of some cardiac disorder.

Such a case as the following is not an infrequent finding in practice:-

> Mr. D. Aged 46 Shopkeeper. Complained of "Feeble feeling" and "heaviness" over the heart in left submammary region. Always complained of being short of breath if he hurried. Is flabby type with neurasthenic background. Was invalided out of army in last war with so called D.A.H. (collapsed while stretcher bearing during training) Complained of frequent attacks of lumbago and sciatica. Now described his pain as "stabbing" character and often lasting whole day. No history of palpitation.
>
> Auscultation systolic murmur audible at apex.
>
> B.P. 120/80
>
> ECG normal except for definite left axis deviation.

The suggestion by Bramwell[27] that the duration of the white tache on the skin is maintained for a shorter time in patients with irritable hearts than in normal controls, suggests here a close association with vasomotor sensitivity. In toxic or infective cases it was found that as the patient's health improved the tache was found to persist longer. It is certainly quite customary in practice to find neuro-circulatory asthenia follow acute infectious conditions or be associated with any septic focus such as teeth, tonsils etc., and quite a few such conditions clear up on the removal of the primary focus of the infection.

As a post-influenzal manifestation it is a frequent occurrence usually disappearing after convalescence.

General Debility. The term general debility is a very useful one to apply and in spite of its vagueness it is not infrequently the only term which can quite describe certain conditions. Such patients frequently complain of a dull ache over the heart although usually submammary and accompanied by a profound sense of fatigue. Usually they describe the pain as occurring late in the afternoon or evening and persisting long after they have gone to bed and this very frequent in women about the menopause and is accompanied by flushing, nervousness or palpitation. The full importance of palpitation as a prime complaint by the patient can be a useful guide to diagnosis, and is more frequently found in association with a cardiac neurosis than organic disease. Palpitation does not always appear to mean irregularity of action and where this term is employed it usually means a precordial throbbing or a general consciousness of throbbing whether that action be regular or irregular. It is therefore a greater consciousness of the heart's action most of all, which appears to be the definition of palpitation to the lay mind. It has always been a matter of great surprise how often we can come across a case of gross cardiac irregularities where the patient

is quite unaware of any alteration of rhythm or rate. Cases of auricular fibrillation or regular and irregular extra systoles have been noted of which the patient was quite oblivious. We know of course that all cardiac irregularities require careful investigation in order to decide which are indicative of organic disease, but where a history of fainting, giddiness and other nervous manifestations is coupled with palpitation as a sympton we are always inclined to suspect a neurosis until proved otherwise. The presence of sighing respiration or some degree of tachypnoea are likewise more suggestive of the benign condition. Radiation of the pain in cardiac neurosis can be extremely confusing since on occasions it can conform to the outline of the anginal syndrome. This however is rare and as a rule a wide and bizarre distribution can be ascertained overlapping the ordinary recognised areas of referred cardiac pain.

The existence of a left submammary pain of general debility is not confined entirely to woman. This type of case is not uncommon to-day.

Mr.W.A. Aged 44 years Railway Employee. Complains of left submammary pain and palpitation on exertion or often at rest in bed. Described this as sharp pain of a stabbing character lasting for a few seconds and followed by a certain amount of aching. Gets frequent palpitation on exertion and while resting in bed. Goes to sleep with hands under head to avoid effects of irregular heart

action. Finds on any slight exertion or with any excitement pulse rate increases considerably. States that he feels exceedingly tired and is working 7 days a week. Long history of neurasthenia following the last war when he was blown up by a land mine. Knee jerks exaggerated. Coarse tremor of hands. Is heavy smoker. This patient periodically has nervous breakdowns although never of a serious nature.

B. P. 140/105

Pulse 80 regular

Auscultation N.A.D. ECG normal.

DISTURBANCE OF RHYTHM.

In this group we have various types of mild or severe cardiac pain associated with either sudden increase or decrease in the heart rate. Peculiarly enough a simple tachycardia seldom produces cardiac pain unless we are dealing with a high rate or with the tachycardia of hyperthyroidism. It is not uncommon however to find various types of left sub-mammary pain associated with a simply tachycardia, but here the tachycardia is only an expression of the general nervous condition of the patient. In hyperthyroidism we do get occasional examples of a substernal pain attributed, no doubt, to one or two factors apart from the mere increase in rate.

In this condition the excessive heart rate produces a greater demand on the part of the myocardium for increased oxygen, but here an increased metabolic rate had already made increased demands on the oxygen capacity of the blood. Likewise a diminished oxygen saturation must result from the increased rate of circulation through the lung thus aggravating an already deficient supply to the myocardium. We have therefore a relative insufficiency established, with, in some cases definite anginal symptoms. Usually however these cases only demonstrate such symptoms on the onset of auricular fibrillation. This type of case is not infrequent

> Mrs T. Aged 26 years. Has complained of palpitation since 1939 following influenza but has always had a large thyroid since school days. Has had several attacks of rapid pulse with "thumping" associated with pain under sternum and under left breast. Was seen during an attack of fibrillation when she complained of left submammary pain on exertion or if tired, sometimes radiating down inside of arm to the elbow. Often the pain persisted all day but would disappear by the morning.
>
> On administering iodine & bromide she improved and when seen recently her pulse was 68 regular and she was free from pain.

As a rule attacks of Paroxysmal Tachycardia are frequently associated with pain often of the anginal type, such as in the following case:-

Mrs H. Aged 44. Rate 120 Regular. Complained of frequent palpitation of two years duration usually after exertion. Following a fall downstairs with child in her arms she had severe attack of palpitation and was admitted to hospital as paroxysmal tachycardia.

Since then has had frequent attacks of "rapid heartbeating" with severe aching under the sternum after her attack. Between the attacks she complained of lack of energy and palpitation on exertion. Occasionally the pain radiated to the back on the left side. This is a highly strung person who suffered considerably during the bombing of London, and dates her condition partly to this.

Auscultation N.A.D. B.P. 150/90 ECG normal.

Decrease in cardiac rate can occasionally produce a pain of coronary insufficiency although by no means invariably the case, and in fact the increased stroke volume as a rule compensates for the slow rate in these cases. Here is a case of Sinus Bradycardia with such a history:-

Mrs L. Aged 45. Complained of lack of energy and great dyspnoea following a nervous breakdown two years ago. When seen recently her pulse rate was 44 and Blood pressure 160/80. Now gets pain left submammary type radiating to the back and often down medial aspect of left arm to the upper forearm on any moderate exertion. Frequently complained of fingers of both hands "going dead".

Pain sometimes came on when she got out of bed, or occurred if she hurried and again often when she had gone to bed if particularly tired.
Sometimes coughing would induce pain, but there seemed to be no one definite factor responsible.

Auscultation no abnormalities.

ECG Sinus Bradycardia

On screening a considerable cardiac enlargement was observed; a not uncommon finding in slow rates.

AORTIC DISEASE.

Pain of a substernal type is often encountered in aortic disease either of a syphilitic or rheumatic origin, and it is not uncommon to find it associated here with the different lesions of the aortic valve. Paroxysms of pain of an anginal type are often experienced and are due in some cases to involvement of the coronary ostia in the disease process or extension of the disease to the coronary arteries themselves. Usually some indication of the existence of aortic disease such as supra sternal pulsation, accentuated heart sounds with appropriate murmurs or alteration in blood pressure readings and development of aortic configuration on X Ray can be ascertained.

Aortic Stenosis. In this condition usually found in the middle aged or old person anginal pain can be an early and sometimes only indication of the disease. The rough aortic systolic murmur propagated into the neck vessels with lowered systolic pressure can be fairly conclusive evidence clinically. The following type of case might illustrate this:-

Mrs P. Aged 72 years. Rate 96 regular. Became conscious of heart three months ago when she began to keep stopping for breath. Then began to experience pain across lower chest on walking. This was confined mostly to left side and disappeared after stopping for a few minutes. Her dyspnoea is becoming progressively worse and the pain now comes on even if moving about the house.

B.P. 110/78

Auscultation musical aortic systolic murmur conducted into neck. Some oedema of ankles. Slight cough. Low [illegible] pulse pressure due to aortic stenosis.

ECG Left bundle branch block of type I.

Here undoubtedly is a case where extensive coronary insufficiency is the result of the spread of aortic disease to the coronary arteries or diminished flow in the coronary circulation as result of a low systolic pressure. This condition has undoubtedly led to extensive and progressive myocardial damage as indicated by the existence of left bundle branch block as seen on electrocardiogram but might be due to a generalised arteriosclerosis or coronary sclerosis. She is now

having pronounced dyspnoea with other signs of failure.

The reverse process can likewise occur in aortic regurgitation where the fall in diastolic pressure can be sufficient to produce impairment of the coronary flow. In those cases of advanced regurgitation it is maintained that only an intermittent coronary circulation exists, and as the result substernal discomfort or pain on exertion is a common and early sign usually associated with some dyspnoea. The following case is such an example:-

Mr. L. Aged 42.years. Milk Dealer. Complained of "gripping pain" across chest as if someone had caught hold of him; coming on usually if walking, but not always appearing with this exertion. Had to stop for few minutes till it passed off. Had tendency to occur if walking at night, but same distance and place will not produce pain in morning or afternoon. Pain of 6 months duration but had been conscious of "knocking of heart" for years, and has become short of breath.

Auscultation. Pronounced diastolic murmur at aortic area and audible left of sternum "3rd interspace"

A relative mitral incompetence was suggested by a presystolic murmur at the apex. Apex beat was displaced downwards but just outside the nipple and was very forcible suggesting left ventricular hypertrophy.

B.P. 270/70 Rate 65

ECG shewed left bundle branch block with notching of QRS complexes in Leads 11 and 111, no doubt resulting from continued hypertension with subsequent ventricular hypertrophy.

In some cases where anginal symptoms and an aortic diastolic murmur are found we are always justified in suspecting a luetic origin.

Here is an advanced case of aortic incompetence with hypertension. The high degree of hypertension presupposes some involvement of the coronary arteries in the arterio sclerotic process supported by definite evidence of advanced myocardial damage in the bundle branch block.

Aortic stenosis associated with hypertension and anginal pain in the younger person can be illustrated by the following case:-

Mrs P. Aged 44. Complained of pain at lower end of sternum on walking or climbing stairs Had to stop each morning on way to station to allow pain to pass off. Onset of pain particularly aggravated in cold weather or going into a cold room or cold bed. This pain was of two years duration and seemed to have developed since her employment in a chemical factory. Very frequently pain would follow her entry into the factory in the morning but was usually associated with effort.

B.P. 170/70 Rate 80 Regular

Auscultation Pronounced aortic systolic murmur conducted to neck vessels.

ECG is virtually normal except for tendency to left axis deviation.

EXTRA CARDIAC CAUSES.

ABDOMINAL CONDITIONS.

In view of the fact that anginal seizures and coronary thrombosis are so frequently associated with some gastro intestinal upset the differentiation of these two conditions from certain abdominal disorders can at times be exceedingly important. On certain occasions in practice one is called upon to discriminate on the spot between a severe attack of angina, a coronary thrombosis, and some other acute abdominal accident. In some cases indeed it may be almost impossible to make an accurate differential diagnosis between say an acute coronary occlusion, and a perforated gastric or duodenal ulcer. We might also include with these such conditions as acute pancreatitis, cholecystitis and perhaps a spastic colon. In cases where this great difficulty does arise it appears to be the accepted principle that a laparotomy should be performed even though the possibility of it being a coronary thrombosis might make such a laparotomy definitely hazardous.

We must remember that the electrocardiographic findings do not always show positive changes immediately following coronary occlusion and therefore such

investigation cannot always be useful. At the same time there are certain broad clinical indications which can be a definite help in attempting to assess the cause in such an emergency. As a general rule in the perforation we can often ascertain some history of previous digestive disorder but we must bear in mind also, the frequency with which anginal pain can be mistaken for indigestion chiefly because of its greater liability to occur after a meal. We know also that the silent ulcer is by no means uncommon, and we must remember the not infrequent association of a true anginal attack of coronary insufficiency with an acute haematemesis owing to the sudden drop in blood pressure although it is suggested that such attacks do not occur with a normal coronary circulation.

Usually however, the pain of the perforation is much more essentially abdominal and the extension is invariably downwards, nor is the same degree of rigidity found in the coronary thrombosis and the vomiting does not simulate that of the acute gastric crisis. The obliteration of liver dullness is usually in sharp distinction to the occasionally palpable liver of the coronary thrombosis. Sometimes however involvement of a pelvic organ has been known to produce thoracic pain but this is rare and the upper organs of the abdomen are always more liable to produce an acute crisis more readily owing to their double innervation, sympathetic and parasympathetic. The pulse and

temperature changes are not valuable aids in the differential diagnosis here but there is no doubt however that in practice a certain experience is developed in the consideration of the facies of the acute abdomen which is never quite simulated in the coronary occlusion. A most useful guide is the extreme suddenness of the abdominal accident and the greater amount of collapse and shock. Very often we will find that the patient with coronary thrombosis has struggled downstairs (if in bed) to make himself a cup of tea, or in some way attempted to alleviate his condition; but in the perforation no such breathing space is possible. In this group of acute surgical complications ureteric calculus can produce similar clinical symptoms, but perhaps the most difficult condition is the acute cholecystitis or empyema of gall bladder. Here the problem is all the more complicated because a definite relationship is supposed to exist between coronary occlusion and cholecystitis, although many authorities doubt it. This relationship is stressed by Rolleston & McNee[28] who emphasise the frequency of radiation to the arms in angina and to the back in gall bladder. Parkinson & Bedford[29] also stress the frequent association between these two conditions but Dr.Parkinson recently stated that he had been unable to find any such relationship, after extensive investigations.

Bramwell maintains that focal sepsis in the gall bladder may affect the myocardium in the same way as focal sepsis in teeth, nasal sinuses or elsewhere, and that many anginal conditions do obtain prompt relief from cholecystectomy. We also know that gallbladder disease predisposes to arterial change of a degenerative nature particularly referable to the coronary arteries and due no doubt to some error in cholesterol metabolism associated with such conditions.

Many simple forms of gastro-intestinal upset can simulate the pain of coronary insufficiency, and are frequently encountered in general practice. In this group we can include a simple gastralgia associated with a flatulent dyspepsia. This can be a particularly complex problem to solve since many of the early cases of anginal pain are not only associated with dyspeptic symptoms but are frequently followed by them. It is not uncommon to find the patient tell us that relief from pain follows eructation even though his condition may actually be anginal in type. Many of the early signs of coronary insufficiency develop more readily with exercise after a meal but likewise a dyspepsia or substernal discomfort may be induced by hurrying or some violent exercise in the same fashion in the normal individual. In these cases the pain is usually associated with the lower end of the sternum although in both forms its may radiate into the arm.

Certain of these cases of so called dyspepsia may be a coronary insufficiency produced by intestinal flatulence forcing up the diaphragm and the heart and diminishing coronary supply by rotation of the heart on its axis. Again the pain of general debility can often be associated with a history of gastric or duodenal ulcer such as in the following case:-

Mrs T. Aged 33 years. Complained some months ago of epigastric pain aggravated by food X Ray investigation showed definite ulcer on lesser curvature. Was treated with rest, diet and aludrox etc. Is now free from her "stomach pain" and is able to get about but feels weak following her period of low diet. Has since developed substernal pain after exertion "quite different from her stomach pain" Describes pain as sharp and seems to "draw the blood from her face". This pain comes on if she is tired after an exertion but not during it. Has a previous history of experiencing a "dead" feeling in the centre of her chest on feeling cold, of some years duration. Often has to stop for shortness of breath but not for the pain. On examination Rate 120 regular.

B.P. 150/100

ECG normal.

I have no doubt that this is a pain due to general debility following her low diet and no direct relation to exertion can be established.

We must remember also that the digestive process by its demand on the splanchnic circulation can thus diminish coronary flow and where excessive exercise follows, a physiological coronary insufficiency may be induced. Conversely where actual coronary sclerosis does exist it may be more readily brought to light by this diminution of coronary circulation after meals. The important point here is that if the patient still has the pain if remaining quiet after a meal or if the pain occurs on exertion with the stomach empty then the condition is more probably anginal.

CARDIO SPASM.

While considering this question of abdominal pains we must bear in mind the similarity to angina of the pain produced by a spasmodic contraction of the oesophagus. It has been suggested by some authorities that all pain of angina pectoris is in reality produced by oesophageal spasm. Morrison & Swalm (31) are quoted as having induced what they believe to be attacks of angina by inflating balloons in the oesophagus and thereby producing distension. These two workers evidently made X Ray investigations of the oesophagus in patients with attacks of substernal pain and found spasm to be a common disorder in the cases studied. The similarity of the pain induced by oesophageal

spasm is very close to that of angina but where the oesophagus is the cause however, a difficulty in swallowing during the attack can usually be discovered, and no relief is obtained from nitrites. They go so far as to state that where spontaneous attacks of anginal pain occur without association with exertion and not relieved by nitrites they should be regarded as oesophageal spasm until proved otherwise. The authors point out the frequency of occurrence of such spasm in nervous individuals, heavy smokers and persons with gall bladder disease; and in this group we might be justified in adding neoplasms. The following case is an example:-

Mr. S. Aged 63 years. Business Man.
Complained of intense substernal pain of one month's duration occurring as soon as he started to eat, and he often had to "stop everything" and "tears would roll down his face". After a minute or two it passed off and he was able to continue the remainder of the meal.
For a short time any attempt at swallowing was intensely difficult during the paroxysm.
Had lost a good deal of weight (1 stone in 5 weeks) and complained of lack of energy.

Examination Auscultation N.A.D.

X Ray findings here shewed some delay with barium swallow and on screening and film large filling defect on greater curvature near cardiac end of stomach.

Radiological diagnosis was neoplasm of the curvature of the stomach. (Lesser)

In this case the similarity of intensity and distribution of the pain to that of angina is very close indeed, but the intense difficulty in swallowing helps considerably in the differentiation.

Other Mediastinal Conditions.

A sternal location of the pain is frequent in subdiaphragmatic pleurisy or pleural effusion or in adhesions in this region and herniation of the diaphragm is another condition productive of similar pain. The pain of acute pulmonary embolism is anginal in type and can be readily confused with that of coronary occlusion. Here the intense dyspnoea, cardiograph changes with probably haemoptysis can be of some help in the diagnosis. The accentuation of the second pulmonic sound can often be elicited although this is by no means diagnostic. A sudden pneumothorax can often simulate greatly the pain of coronary occlusion, but here the electrocardiogram can be diagnostic in conjunction with X Ray findings.

The persisting ache of hypertension is another confusing condition and although induced or aggravated by exertion it is considerably less severe in type. The following case is such an example:-

Mrs M. Aged 66 years. Complained of ache across lower chest usually after exertion, and of some years duration. This was by no means constant or severe. Three years ago had colporraphy performed which was followed by acute arthritis

in arms leg and neck. This had now subsided but blood pressure was 240/120 and she complained only of pruritis and chest pain. Seen recently with pulse of 90 regular, fundi vessels wiry, discs clear. Blood urea 86.6 Urine negative for albumin but sp. gr. 1010 in all specimens. (Arteriosclerotic kidney).

Auscultation accentuation of first mitral sound accentuation 2nd aortic with soft systolic murmur.

ECG normal.

Substernal thyroid is another condition which either from pressure or the tachycardia can produce a certain amount of substernal discomfort. The neck fullness on swallowing with head forward and raised, and the wedge shaped shadow under the sternum on X Ray coupled with tremor etc. can be almost diagnostic.

Diseases of Chest Wall.

These are the most frequent clauses of precordial pain encountered in general practice, and we could put first general strain of the pectoral muscle. Myalgias of the pectoral muscles and various rheumatic conditions would naturally include rheumatism of the sternal articulations and these one might say come next in frequency. Here the association of the pain with movement and tenderness of the muscles on deep pressure with the muscles on stretch is often fairly diagnostic.

Myofibrositis and even an early mastitis can occasionally be confused although in the former the pain is usually worse on getting up in the morning and passes off as the day goes on.

Intercostal neuritis or neuralgia can often be extremely confusing and one of the American Journals quotes an instance of a doctor who was convinced that he had angina until the eruption of herpes clarified the whole condition. Rare conditions such as various infections of the sternum or bony parts of the thorax such as osteomyelitis, periostitis or neoplasm can produce pain similar in type.

Nerve Root Pains.

In certain rare cases radiation of anginal pain can be found extending into the left arm and fingers, without any substernal or cardiac distribution. This should always be borne in mind when a diagnosis of brachial neuritis is being made, and it is worth enquiring into any association of an arm or neck pain with exertion. Cervical arthritis can produce this type of pain usually in the deltoid and trapezius muscles as the result of involvement of C4, C5 and C6. Again we may also have pain in these muscles referred from a diaphragmatic pleurisy by way of the Phrenic nerve into the fourth, 3rd and even the 5th cervical nerves.

Usually however, the characteristic spread of the anginal pain is limited to a relatively small territory of the left anterior axilla supplied by the 2nd thoracic nerve and the inner aspect of the left upper arm and forearm down to the central aspect of the little finger supplied by the 2nd and 1st thoracic nerves forming the "ulner distribution". Pain in the left arm from other causes is usually more extensive and overlaps the areas of the musculo cutaneous, the radial, the musculo spiral or the median nerve as well as the ulner nerve.

Neuritis of the left cervical and the brachial plexus can follow fractures or dislocations of the shoulder or may result from pressure on the plexus of tumour or vertebral caries. The association of brachial neuritis with post influenzal conditions, toxaemias, anaemias or vitamin deficiencies is well known. Sometimes neuritis of these lower cervical nerves will involve the sympathetic trunk producing Horner's syndrome with contraction of the pupil narrowing of the palpebral fissure, partial loss of accommodation to light and other vasomotor changes in the neck and chest. Where this condition is associated with injury to the cervical sympathetic nerve it is classed as Klumpke's paralysis. Cervical rib is another cause of unilateral neuralgia and may involve either the cervical or brachial plexus. Occasionally spasm of the scalenus anticus can be found in association with

chest pain such as in the following case:-

Mr. G.E.B. Aged 58. Engineer. Complains of shortness of breath 3 months duration with frequent palpitation. Has recently had occasional attacks of substernal pain radiating to both sides of neck with sudden twisting of head to the right. This usually lasts a few minutes then passes off occurring often about once or twice a week. It appears to have no relation to exertion.

Auscultation N.A.D.

B.P. 120/65 Rate 76 Regular

ECG normal but pronounced Left axis Deviation.

This case was regarded as a possible bizarre distribution of anginal pain but subsequent observation appeared to negative such a conclusion but negative findings are not conclusive in this type of case.

In pain due to pressure from a cervical rib relief can often be obtained by trying pressure of the hand behind the neck in such a way as to push the cervical rib forwards from the cervical plexus. X Ray investigation is usually definite here.

Syringo-myelia can produce a neuritis of left cervical distribution, but dissociated anaesthesia i.e. loss of temperature and pain sense with persisting tactile sense is usually diagnostic.

Spondylitis of the upper thoracic or cervical vertebrae can be exceedingly confusing since this condition can be aggravated by exertion or unfavourable posture and is relieved by immobilisation. Cord lesions may or may not cause neuritic pain but usually the pain here is bilateral with some spastic signs below the lesion. It is not infrequent to find anginal pain radiate to the face, neck and head, and it is suggested by some writers that these pains may be cardio-aortic in origin and that the sensory portion of the 5th and 10th nerves may carry the pain.

TOXIC FACTORS.

Various toxic factors can, by their effect on the nervous system produce pain in the heart which may simulate very closely the pain of angina. Tobacco is a great offender in this respect although there seems to be great difference of opinion as to whether an actual angina can be produced by it, but certain people are definitely susceptible to something in tobacco which can produce considerable cardiac discomfort. Very often the discomfort involves the hypochondrium with some chest pain, pallor and sweating. Herrman[32] maintains that some substance in the tobacco does restrict coronary flow in people susceptible and quotes experiments showing frequency of positive patch tests with tobacco in cases of coronary

disease. There is no doubt that some association appears to exist and many writers have tried to prove an association between tobacco and the occurrence of coronary thrombosis, but the most recent opinions seem to cast considerable doubt on this association.

Coffee is another frequent offender, and Levy[33] maintains that anginal attacks from caffein are comparatively frequent and can be found to occur at rest and clear up entirely on giving up coffee. Adrenalin and Insulin are both capable of producing paroxysms of anginal pain and an attack is not uncommon on the sudden development of diabetic coma. Gout and diabetes are two conditions where neuritis is a common complication but we must also bear in mind the frequency of coronary disease in these two conditions, it being suggested by some writers that a high percentage of diabetics subsequently develop coronary thrombosis. Menopausal conditions, particularly where associated with a hypochromic anaemia and increase vasomotor sensitivity can produce many types of heart pain but usually of the submammary type.

Miller[34] suggests that many cases of vague abdominal conditions where in despair surgical treatment such as removal of appendix or gallbladder is resorted to only to find their pain continue are probably anginal with an aberrant distribution. Here is an example of abdominal pain:-

Mrs C. Aged 63 B.P. 180/100
Has complained of pain in the right hypochondrium for the past 6-8 weeks only coming on during exertion such as walking etc. and relieved in a few minutes with rest. Dyspnoea increasing recently cum some oedema of ankles. Has had palpitation or fluttering of heart for many years.

Auscultation no abnormality, response to nitrites almost immediate.

ECG shows left bundle branch block.

ATYPICAL ANGINAL PAIN.

Before concluding this chapter we must bear in mind that certain rare cases of peculiar distribution of the pain do occur in angina. These have been recognised by many writers and appear to be in two separate groups (1) The eccentrically placed pain and (2) the chronic left shoulder pain.

1. Eccentric Pain.

Various cases are recognised where the pain seems to involve the arms only but still has the same relation to exertion as the centrally placed pain of angina.
J. Spillane & Paul White[35] summarise these findings in their article in the British Heart Journal and suggest

the possibility of an ischaemia of the forearm muscles. It is a very interesting suggestion although the exact mechanism appears to be unknown. Robertson & Katz, 1938 (34) are quoted in this same article as having induced paroxysms of anginal pain in 19 out of 24 anginal patients by restriction of circulation in the arm for 5 minutes at a pressure of 50 m.m. above the systolic pressure. Their results suggest that by producing one element of the spontaneous attack the paroxysm can likewise be induced in its entirety. The interesting feature of these cases is that on many occasions this is the only form which the anginal seizures take, often for a period of years before the true anginal paroxysm develops.

2. Persistent Shoulder Pain.

Classical Angina Pectoris or infarction is occasionally complicated by this shoulder pain. In this series several cases were found where a neck or shoulder pain was present sometimes for a week or two prior to a myocardial infarction. Some cases are on record where such pains are present for many weeks or even years. This pain has no relation to exercise and is not relieved by trinitrin. Some American Writers describe a wrist pain as a forerunner of an anginal seizure.

This peripheral location of the pain prior to its central development is not infrequently found and

may be illustrated by the following case:-

Mr.C. Aged 66 years. A Cellarman. Had complained of a substernal pain while walking or doing any moderate exercise. This condition had been present for 8-9 years but had become much more pronounced since he came to Harrow from Margate two years ago. He found that a distance of 50 yards walking brought on his pain and he had to stop to get relief. Of recent months even coughing at night or attempting to do any gardening would likewise produce discomfort.

The interesting feature in this case was the occurrence of the pain along the lateral aspect of both thighs before and after the substernal pain. On some occasions discomfort persisted in the thighs for some hours after his attack.
Dyspnoea is becoming a pronounced feature particularly the last 12 months.

Auscultation Aortic diastolic murmur with suspicion of gallop rhythm.

B.P. 150/80 Rate 80 regular.

ECG shews low voltage in classical leads with high P2 and P3,T3 is diphasic.

Here a high degree of depression of the ST segment in leads 1 & 11 is strongly suggestive of some chronic coronary insufficiency. The high pointed P2 & P3 support a diagnosis of auricular enlargement - no doubt part of general cardiac enlargement. (P 172)

ARTERIOSCLEROSIS.

In making any approach to the study of coronary disease and its effects on the myocardium we are, in most cases, discussing the effects of anteriosclerosis generally and of the coronary arteries in particular. We know that all cases of coronary sclerosis are not always associated with general arteriosclerosis but it is generally recognised that it is above all things the most frequent occurrence. Paul White (35) illustrates the results of his investigation into a series of cases of coronary insufficiency and shews that in his experience nearly every case of angina was found to have arteriosclerosis of the coronary arteries and particularly the left anterior descending branch as the principal finding. His exceptions were

1. Athero sclerosis - a few cases
2. Luetic aortitis involving the ostia often without involvement of the artery - a few cases
3. Extreme rheumatic regurgitation in young persons - a few cases
4. Sub acute bacterial endocarditis with vegetations involving the coronaries - one case
5. Congenital anomaly - one case

It is obvious from the above findings that coronary arteriosclerosis is a frequent finding and in White's

opinion it occurred in over 90% of the cases investigated by him. In Glasgow in 1000 necropsis Allan found a coronary lesion trifling or severe in 37.1% visible to naked eye alone (Levy).

It is suggested by most authorities that diabetes glomerulo nephritis and hyperthycoidism are frequently found in association with this condition. The suggestion that the present hurry and bustle of modern life with its great emotional stress may have some etiological association with arterio sclerosis is rather contradicted by Levy[36] who points out the existence of this condition in mummied corpses of the Egyptians who lived 3000 odd years ago. A definite association with occupation however seems to be established although contrary to expectation the professional and executive classes do not head the list although they come very high in this order. In Levy's[36] investigation the foreman and skilled worker class seem to suffer most readily from this condition. The association of hypertension with arterio sclerosis is of course common and there seems every reason to believe that continued hypertension favours degenerative changes in the arteries. It seems that the effect of angiotonin of renal origin may be due, not entirely to its vasoconstricting action but also to a stimulation of the myocardium with increase in the volume of ventricular systoles. And it is suggested by Philip Hill & Cowes Andrew[37] that here may be one of the

important causes of arterial hypertension. The whole question of heredity is an important one, and there seems little doubt that this ia a strong factor in the occurrence of arterio sclerosis and hypertension.

THE ELECTROCARDIOGRAPHIC INVESTIGATION.

In the investigation of cases with a history suggestive of coronary insufficiency we have three separate sources of information viz: the clinical findings, electrocardiographic findings and X Ray findings, and the relative significance of these is the object of this investigation.

The first, and in most cases the most important source of information is the clinical findings. In many cases these findings can be sufficiently diagnostic in themselves, particularly where the distribution and occurrence of the pain conform to the recognised form. This is naturally simpler in the more advanced cases of anginal pain, but there are, unfortunately, still many cases where the clinical findings are by no means unequivocal especially where the symptoms are of comparatively short duration. This grouping does not, of course, include those cases where a recent but definite clinical history of coronary thrombosis can be ascertained.

When we are faced with a case giving no definite or conclusive clinical findings we must perforce rely more on the second and third sources of information, namely the findings of electrocardiography and radiocardioscopy.

The latter two sources of information are, after all, merely subsidiary means of gaining further knowledge of the patient's condition but in no wise should be considered apart from the clinical background.

The electrocardiogram, if interpreted cautiously and with due reference to the patient's history can often give us additional information which at times can be very conclusive, but we must bear in mind that there is no typical electrocardiogram of coronary arteriosclerosis. The electrocardiogram gives us a picture of the state of the myocardium and since this can be influenced by many conditions other than the circulation of the coronary arteries, care must be exercised in the interpretation of such records. Various anaemias, fevers, toxaemias, drugs etc. can produce changes in the myocardium and hence in electrocardiogram and with this in mind, we must therefore be careful in attempting to make any sweeping statement regarding the state of the coronary circulation without due consideration of all information from other sources. At the same time we find that certain electrocardiographic changes are more frequently associated with coronary

disease than others, and in using the electrocardiogram we must try to assess first what particular changes we are prepared to consider as diagnostic features of value. In order of importance the following findings can be useful in support of a doubtful clinical history:-

(1) Signs of recent myocardial infarction

(2) Signs of healing myocardial infarction

(3) Signs of old healed myocardial infarction

(4) Signs of myocardial damage or necrosis and repair of a chronic nature

(5) Signs of deficient coronary supply and its effect on the myocardium showing as permanent changes or slowly evolving changes

(6) Signs of affection of auricles only in coronary insufficiency

(7) Signs appearing only on induced anoxaemias

(8) Signs appearing only after exercise, but with return to normal on rest

This grouping is a most comprehensive one and can include nearly all possible electrocardiographic changes, since it is possible for coronary insufficiency to produce almost any form of deviation from the normal. Without however going into what might be called the very fine points of this science, we can at least hope to consider what main changes we can accept and the relative significance of such changes.

There have been so many publications on this subject and some authors are so prone to read into the electrocardiogram what is still a matter of doubt, that we must adhere to what we can regard as diagnostic aids without the consideration of changes which are in the present state of our knowledge unreliable. In fact it seems that in the past a greal deal of harm has been done to the progress of this science by rash pronouncements on minor changes in the elextrocardiogram.

In the first group, namely those cases showing signs of recent myocardial infarction, the electrocardiogram can be of definite help.

This however is by no means always the case, and a record made too e arly (say 24 hours) can sometimes miss the development of such changes.

In a minor infarct, or where the area involved does not happen to be in a part of the heart which shews readily, the changes can be extremely transient - a few days at most, with complete and rapid return to normal. This, we must admit, is the exception, but in all cases it is always advisable to have a follow up record made within a few days since it is the process of evolution of the electrocardiogram in myocardial infarct which is one of its diagnostic features. The typical changes vary in the time of their occurrence but generally appear from about twenty four hours after the incident, to something like ten or twelve days.

The duration and extent of the changes depend so much on the location and extent of the infarct and the extent of the collateral supply; the latter factor being perhaps the most dominating factor of all.

It is not uncommon for autopsy findings to shew occlusion of one or both coronary arteries where no incident suggestive of an acute coronary accident was recorded during the patient's lifetime. These cases are frequently found to have considerable collateral supply and to be almost independent of the normal coronary circulation.

The Coronary Electrocardiogram.

The part of the curve normally most affected by this condition is the ST segment and it rapidly undergoes significant changes in elevation or depression above or below the base line. This change in the shape of the ST segment is of the greatest importance and in these cases the segment usually passes to the top of T by a straight line or by a curve which is concave towards the base line in the elevated segment.

It seems to be the theory accepted by recent authorities such as Pardee and Katz and others that the ST changes are the reaction to degeneration or necrosis of the myocardium produced by the myocardial ischaemia. The ST segment changes represent the current of injury and are understood

to be produced by changes at the junction of the necrotic tissue with healthy tissue. Tissue which has been totally destroyed does not produce such a current and it is for this reason that we must be careful in interpreting a return to normal on the part of this segment as signifying that recovery has taken place.

It might be that necrotic tissue has been replaced by fibrous tissue which has no electrical reaction and therefore no such current of injury is produced. The whole approach to the interpretation of the electrocardiogram in such cases is simplified if we consider the elements which go to make up this so called "coronary electrocardiogram" bearing in mind, of course, the fact that wide variations are possible and also that certain other conditions can give such a record. The elements of the coronary electrocardiogram consist of certain changes in the ST segment and in the T wave. The ST segment changes consist of either elevation or depression of the segment above or below the isoelectric line but as already stated, the actual shape of the segment is of supreme importance.

This characteristic shape of the ST segment consists of a filling in of the normal upward or downward concavity before the peak of the T wave, so that the line runs either in a straight line to the peak of T or is slightly bowed. This is usually associated with a displacement of the segment beyond normal limits in the direction of

the T wave. Advanced forms of left or right axis deviation can shew somewhat similar ST changes but here the RT junction is usually below zero level or above in the opposite lead, and the ST segment has its normal concave appearance. This is a particularly important point and arises later on in the discussion.

Corresponding T wave changes producing the so called "coronary T wave" are usually found associated with such ST changes in infarction. The chief feature of this wave is a slight upward convex shoulder and then an inverted T wave with symmetrical sides and sharp point. The important feature is the slight upward rise above the point of origin prior to the down stroke of the T wave. This wave may, or may not, be diphasic. It is possible for certain other conditions to give a coronary T wave but in most cases it is due to coronary disease. The apex of the T wave is usually downwards and may be accompanied by downward T in other limb leads although these T waves may not be of coronary types. If such changes occur in Lead 111 only then the diagnostic value is slight, but if accompanied by T inversion (not necessarily coronary T) in Lead 11 or an isoelectric T2 it is much more significant. The coronary T wave is found in the precordial leads during healing stages of anterior infarct but not when the diaphragmatic surface is involved unless accompanied by anterior infarction (Pardee)

Such then are the characteristic changes of the coronary ST and T waves and these groupings appear in greater or lesser degrees in many electrocardiograms associated with coronary disease.

By far the most definite changes associated with cardiac pain are those of the acute myocardial infarct. Here as a rule the clinical findings can be fairly suggestive but this is by no means always the case, and where any doubt exists that an acute coronary accident may have occurred the electrocardiogram can often be very decisive.

The changes occurring in infarction can be tabulated as follows:-

Recent Effects.

ST segment elevations or depressions with characteristic concavity or convexity of the segments. Of these the anterior and posterior infarctions have their characteristic changes.

Anterior left ventricular infarct.

Early.
1. Convex elevation of ST segment Leads 1 and lV
2. Concave depression of ST segment Lead lll
3. May or may not be Q wave in Lead 1
4. R4 may or may not disappear

Later.
1. ST segments tend to return to normal
2. T waves dip late, sometimes inverted Leads 1 and lV and even in Lead ll
3. R4 usually absent

Basal or Posterior or Diaphragmatic Infarction.

Early.

1. Elevation St segment Lead 111
2. Depression ST segment Lead 1 and
3. After hours or days Lead 1 and IV return to normal or have too high waves
4. Q waves in Lead 111 with sharp inversion of T waves in Lead 111

Later.

1. ECG tends to return to normal
2. Q3 and inverted T3 only remain.

Late changes.

In healed cases after many months all evidence of the infarction may disappear except for the following changes:-

Anterior Group.

1. Absence of R4
2. T1 and T4 often remain inverted or diphasic or flat

Posterior Group.

1. ECG tends to return to normal
2. Q3 and T3 changes often remain the only evidence.

It is usual for Lead 11 to simulate the lead shewing the elevated segment only to a lesser degree, but unfortunately such definite characteristic changes only appear in a certain proportion of cases. Many atypical forms of this pattern are found and in some cases patterns of combined infarcts are found.

When we are dealing with the elevated ST1 segment I type of case with the later development of the coronary T wave in this lead, it is common to find a broad Q wave develop in this lead with a deep S wave Leads 11 and 111 This has given rise to the term Q1 T1 type of anterior infarction.

In the reverse pattern with coronary T wave in Lead 111 the Q wave usually develops in this lead. Frequently the QRS consists solely of a wide downward deflection or QS in the third lead. This has given this group the term Q3 T3 pattern. The QRS changes are more permanent and hence these Q waves are often the only remaining evidence of an old infarction.

The chest leads usually simulate those of Lead 1 and in the anterior infarction the chest leads usually (though not invariably) give us most information.

In the posterior infarction the chest leads often give little information and may be quite normal. It is not uncommon to find a large pointed T wave in the chest leads with a healing infarction and this is often regarded as a significant feature on investigation of cardiac pain. It is however, only to be regarded as such if it is found to undergo definite process of evolution.

The disappearance of the R wave in the chest leads is of great significance and is one of the remaining signs of myocardial infarction, especially if taken from the

apex where R is usually large.

In the posterior or diaphragmatic infarction the R wave is usually present because the degenerated portion of the muscle is not immediately below the electrode. The importance of such definite findings in our investigation cannot be overestimated, since it is not uncommon for myocardial infarction to be atypical clinically. The prolongation of myocardial ischaemia from whatever cause can produce infarction and therefore a typical history with the characteristic location and radiation of pain is by no means the rule. It is common to find a history of angina of effort follow myocardial infarction, and some authorities go so far as to state that 65% of cases of angina of effort have a myocardial infarction as the initiating factor. (Rosenbaum & Lavine)(14)

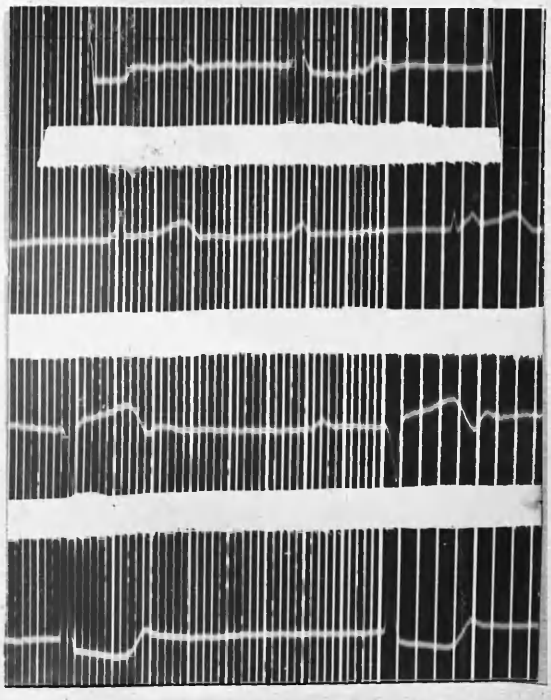
Indirect cardiac trauma producing a prolonged myocardial ischaemia with a limited infarct is a frequent occurrence. This may be due to an upset of the balance of coronary supply and myocardial need, but might be due also to some indirect injury to the coronary wall such as a petechial haemorrhage into the intima. It is suggested by other writers that this infarction follows exercise (Jokl & Susman)(43) and is due to a physiological drop in blood pressure following the rise with exertion. This would seem to apply only to cases where actual limitation of coronary supply already existed or where considerable

myocardial hypertrophy was present.

It has always seemed that the type of case with infarction occurring after prolonged exercise has a better outlook than where infarction occurs during rest since probably a greater limitation of coronary circulation is implied by the latter.

The typical infarction usually does occur at rest and such is the following example.

Mr.A. Business man - aged 68 yrs



Seen 24 hours after attack of intense substernal pain with no radiation occurring during the night lasting about 30 minutes followed by considerable discomfort for many hours. History of some substernal pain on exertion (moderate) at infrequent intervals often after meals.

B.P (now) 160/100 No other abnormal signs but pulse rate found to be 40, a history of hypertension was obtained.

The ECG shows characteristic depression of ST1 and ST4 with elevation of ST2 and ST3. with developing inversion of T3 with prominent Q3, indicative of Posterior Infarction.

B.P (was) 185/

This patient was under the impression that his condition was due to indigestion and indeed he had already made up his mind as to its causation.

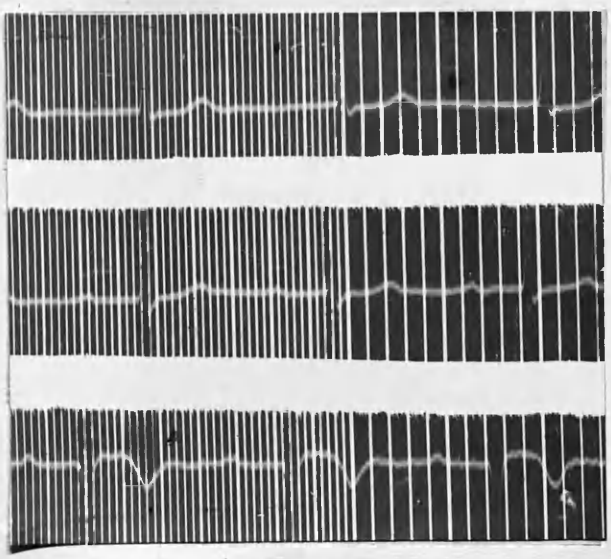
The electrocardiogram here was very helpful and typical changes characteristic of the posterior or diaphragmatic infarction were evident with an associated rhythm disorder. In this case a complete heart block with regular ventricular rate of 40 and auricular rate about 80; was observed.

The associated disorder of rhythm is a common feature of the posterior infarction since the blood supply to the septum is usually involved and hence the conduction system frequently shows some impairment. Various rhythm disorders are a frequent accompaniment of the posterior infarction partly because of involvement of the septum and partly due to the fact that the conduction system as a whole is much more susceptible to ischaemia than the heart muscle. Various grades of conduction defect can appear either as a transient or permanent feature following infarction. These include such disorders as the above with varying degrees of A V block and occasionally bundle branch block or incomplete bundle branch block.

This case then seems to illustrate not only the typical early ST changes of posterior infarction but also associated rhythm disorders all of which can be helpful in diagnosis of an atypical clinical history. In fact in this case the history was so uncharacteristic that, but for the electrocardiogram,it might have been missed,

since by the next week or two the pulse had reverted to normal rhythm and an electrocardiogram shewed a further step in the evolution of this type of change namely normal rhythm with prolonged AV conduction time (.32 seconds)

Here the ST changes are returning to normal with the development of a pronounced coronary T wave in Lead lll with still prominent Q3. A lower grade of heart block has now developed and is shewn as prolongation of AV conduction only with long PR interval, but each P wave followed by a ventricular wave

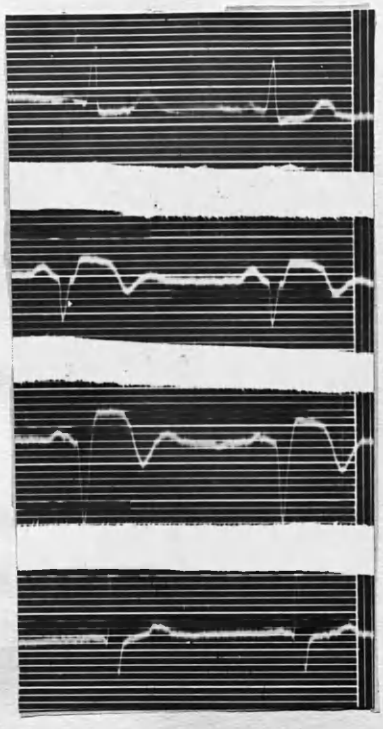


Such a record suggests involvement of the base of the septum

This illustrated the importance of the electrocardiogram in any case of sudden appearance of a rhythm disorder, since heart blocks, auricular fibrillation or flutter and even multiple ectopic beats can result from a coronary thrombosis.

A very frequent finding in this series has been the occurrence of a prodromal period of anginal pain preceding a definite clinical attack of coronary thrombosis and it is important that any sudden attack of anginal pain should be treated as an incipient infarction and bedrest insisted upon at once. Sometimes such a minor precursor of the major attack may be an actual coronary thrombosis and

may not always involve the branches or myocardial area involved in a subsequent attack. This prodromal period of aggravated pain often extends for several days but unfortunately the patient does not always report until the major accident occurs. The following case might illustrate this point:-



Mr.W. Aged 63. Retired business man was suddenly seized during the night with substernal pain radiating to posterior aspect of both arms as far as the elbow. He gave a history of substernal discomfort which he thought to be indigestion for 2 days prior to his attack. A history of hypertension was obtained although when the record was taken his B.P. was 120/100

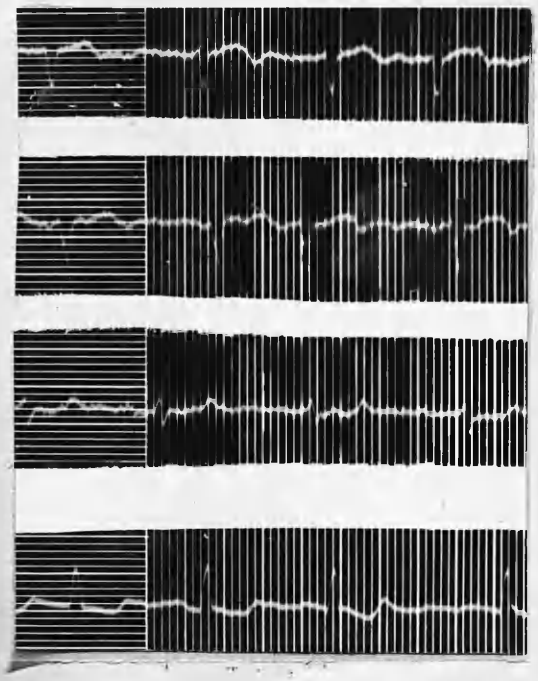
On examination no other abnormalities were found

The Electrocardiogram shewed a rapidly evolving pattern of posterior infarction with already (48 hours) sharply developing coronary T wave in Leads 11 and 111.

This shews the typical ST elevation in Leads 11 and 111 and depression in Lead 1, with associated Q3 and Q2. The chest lead here is normal. This patient subsequently developed a left sided hemiplegia probably thrombotic or embolic in origin. Perhaps the drop in systolic pressure may account for the occurrence of such thrombotic accidents following a coronary thrombosis and it is an important point that many such cases not only shew a drop in blood

pressure at the time, but often continue to show low blood pressure reading sometimes even permanently. This patient made a good recovery but with some degree of residual anginal pain on even moderate exertion.

It has always been a matter for speculation whether any particular type of infarction is more prone to leave residual discomfort on effort but it has not been my experience to establish any such relationship; and it seems that the extent of the primary infarct and probably the degree of collateral circulation must be the governing factors. In this consideration we must include of course the existence of any other indication of myocardial damage. Where such findings obtain then of course the outlook is less hopeful. The following case is an example of a coronary thrombosis superimposed on an auricular fibrillation, an uncommon occurrence.



Mrs B. Aged 50 Admitted to Hospital complaining of sub-sternal pain radiating to left arm. This was a known case of fibrillation and had had some pain on exertion

The electrocardiogram here shews elevation of ST1 and ST2 with depression of ST3 and 4. **Right axis-deviation is evident.**

Here a prominent Q1 and Q2 obtain. The low voltage may be due to the state of her myocardium following the infarct.

Subsequent history on discharge was not obtained.

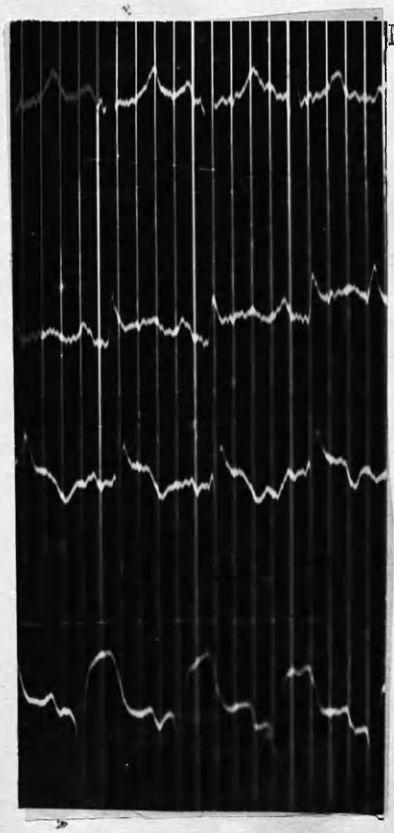
These cases serve to illustrate the more characteristic findings of myocardial infarct and where such findings are obtained the electrocardiogram can be of decisive value, even in a case with little or no clinical findings of a characteristic nature.

The matter however becomes much more complex when we try to assess the value of the atypical changes in the electrocardiogram, which unfortunately can be many, and where a combination of different infarctions superimposed can neutralise some of the diagnostic features of the record. In some cases prolonged anginal pain preceding a major attack may be of sufficient severity or duration to produce some myocardial damage in other areas. In some cases the infarction may be a simultaneous involvement of the anterior and diaphragmatic surfaces of the heart.

In the recent stage of such combined infarctions the ST segment is frequently elevated in all 3 leads although usually most marked in Lead II. In such records a large Q3 may be present and in the precordial leads the R wave is small or absent with some elevation of the ST segment. In the healing stage these cases may present a normal record in the limb leads with an absent R or diaphasic T wave in the chest leads. Such records are liable to be confused with a pericarditis but here no Q wave develops and usually the ST changes are most pronounced in Lead I.

An infarction of the lateral wall of the left ventricle usually produces a depression of ST 1 and ST 2, but no elevation of ST 3 and a depression of the ST segment in the chest leads. This type of infarct is prone to undergo a very rapid process of evolution and can, at times, closely simulate the record of hypertension with T1 changes. The combined infarction most frequently shows the posterior changes occurring in the limb leads with the anterior changes appearing in the chest leads and, I believe Katz (65) Page 182, states that the reverse is exceedingly rare.

The following record is an illustration of this type:-



Mrs G. Aged 57 years. Seen complaining of sudden attack of supramammary pain occurring in bed at night, with no radiation. Here a definite prodromal period of severe pain in a similar location was observed for several days prior to her attack.

Seen 48 hours after her severe attack which had persisted for several hours and which was relieved by morphia

On examination split first sound with pronounced accentuation of second aortic sound were the only abnormalities.

B.P. was then 210/100

The electrocardiogram here shewed definite elevation of ST 2 and ST3 with tendency to inversion in T3 and some depression of ST1 also Q3

Here are typical posterior changes in the limb leads but in the chest lead we find definite ST elevation and absent R wave characteristic of the anterior infarction. It seems reasonable to assume that such changes occurred comparatively recently although it is not uncommon to find a recent infarction superimposed upon the record of an old healed infarct.

Apart from the above recognisable types of unusual change certain atypical forms can appear which do not fit in into any particular group. Such changes may involve the ST segment and T wave in a minor form, but all changes due to infarction have one important diagnostic feature namely their process of evolution, usually with a subsequent coronary T wave. Less frequently R1 and R3 may become smaller and Q1 or Q3 may become larger yet no characteristic feature may develop. Such abnormal changes may be due to the fact that the infarct is small or involves an area not coinciding with the usual areas of infarction. Some authors classify these changes and Katz described a TN and Tp type where coronary curves exist but where ST and T wave changes are the same in Leads 1 and in lead 111. In the Tn type an elevated ST contour is present in all limb leads and in the late stages an inverted T is present in all limb leads. Sometimes a Q wave may be present and the chest leads are variable. In the Tp or positive T types the reverse occurs but this is very rare.

These changes therefore can be of diagnostic value in the study of cardiac pain but their value diminishes the further they become removed from the orthodox changes, and it must be remembered that there are many causes other than coronary sclerosis which can produce some of these changes.

Voltage of QRS and T. Waves.

The mere presence of low voltage QRS is no definite indication of myocardial disease since it can be produced by such conditions as diabetes, myxoedema general debility and others but it is nevertheless an important finding in an investigation of this sort. Pardee (66) (page 172) quoting Barratt (?) states that when low voltage of the QRS was found in combination with the low voltage of the T wave 92% (in his series) had evidence of cardiac disease. He emphasises however other causes of such low voltage T waves of an extracardiac nature.

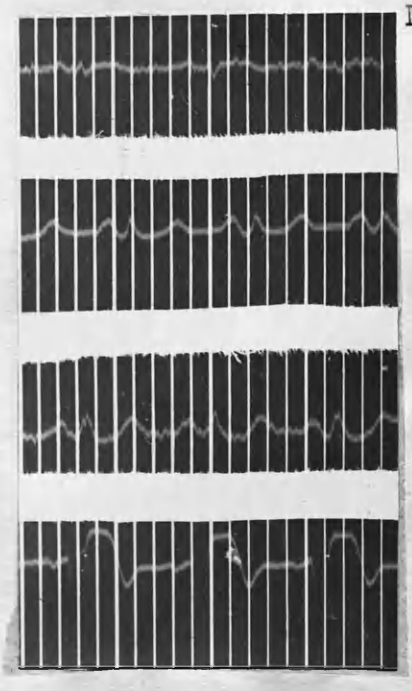
The presence of large T waves may occur in myocardial hypertrophy or in coronary occlusion or in lesions of the bundle branches. A small T wave on the other hand should not of itself be regarded as a definite indication of myocardial damage except in lead I but should always be considered in relation to the QRS wave and where this also is small the cause may be of a toxic or metabolic origin.

If we find a large or normal QRS associated with a small T wave then we can assume a greater probability of myocardial

disease. If, on the other hand significant notching of the QRS occurs, the tendency for a large T wave is very great and not even myocardial disease can produce a small T in such an arrangement. Pardee (66) (page 173)

The sudden appearance of low voltage of QRS and T wave is frequently the result of a myocardial infarct and certainly should call for a careful investigation of the clinical history in such a case. It should be further remembered that in some cases a low voltage can remain as a permanent feature following a myocardial infarct particularly if that has been extensive.

The following case is an illustration of such low voltage changes occurring immediately after myocardial infarction -

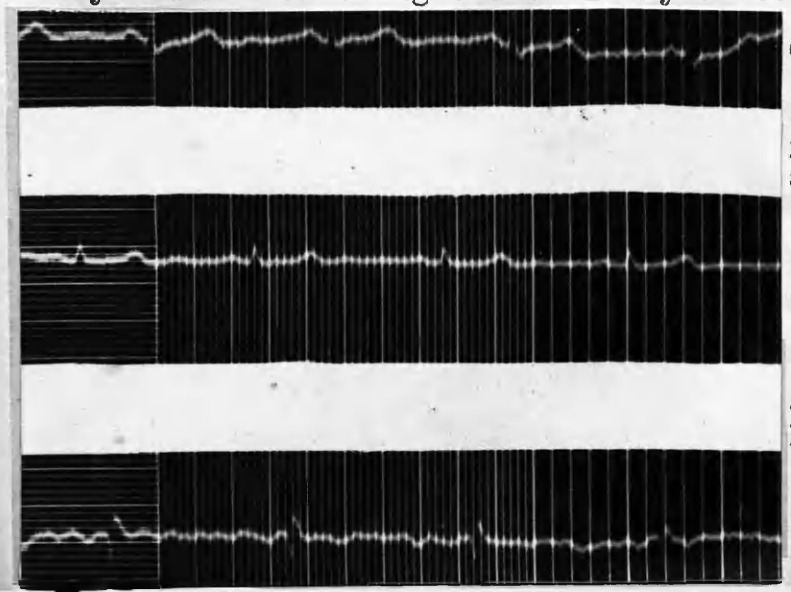


Mr. J.J.G. Aged 67.
This man had a history of occasional attacks of pain in left mid axillary line occurring on strenuous exercise such as hurrying for a train but not with normal exertion.
Was seen two days after an attack of intense substernal pain radiating along ulnar aspect of left arm to elbow and persisting for 12 hours but relieved by morphia.
Here the story is complicated by a previous history of gastric ulcer with haematemesis
The possibility of a perforated ulcer had to be considered particularly as the attack was associated with vomiting.

As already discussed the characteristic pain and localisation in the chest were of great assistance. A history of hypertension was obtained but when seen the B.P. was 130/75.

In this case the ECG was of diagnostic importance and shewed the changes of an anterior infarction with extreme low voltage in the limb leads. The characteristic changes were most pronounced in the chest lead where the absent R wave and ST elevation with developing coronary T wave were evident. The occurrence of low voltage in diabetes with coronary disease can sometimes lead to difficulty, these two conditions being unfortunately associated together and not infrequently such diabetics experience some degree of cardiac pain. This applies equally to myxoedema and here not infrequently similar conditions obtain.

The following case is a record taken from a case of myxoedema following subtotal thyroidectomy two years before:-



This patient gets a considerable degree of substernal pain on moderate exertion although this condition is improving clinically on small doses of thyroid but as yet no improvement in reduction of general cardiac enlargement appears on screening. ECG shows low amplitude of QRS and T waves.

Other possible factors in the production of low voltage can be of a purely mechanical nature such as depression of the base of the heart by mediastinal tumour or upward pressure of the diaphragm from various abdominal conditions producing a transverse lie of the heart.

On the other hand any general condition producing impairment of the physiological state of the myocardium can result in lowered QRS voltage and although coronary sclerosis is perhaps the greatest offender in this respect we must remember that such conditions as tuberculosis, rheumatic fever, anaemia, pericardial effusions and infectious diseases can produce a similar condition by impairing the coronary flow or in the case of pericarditis by the insulating effect of the effusion.

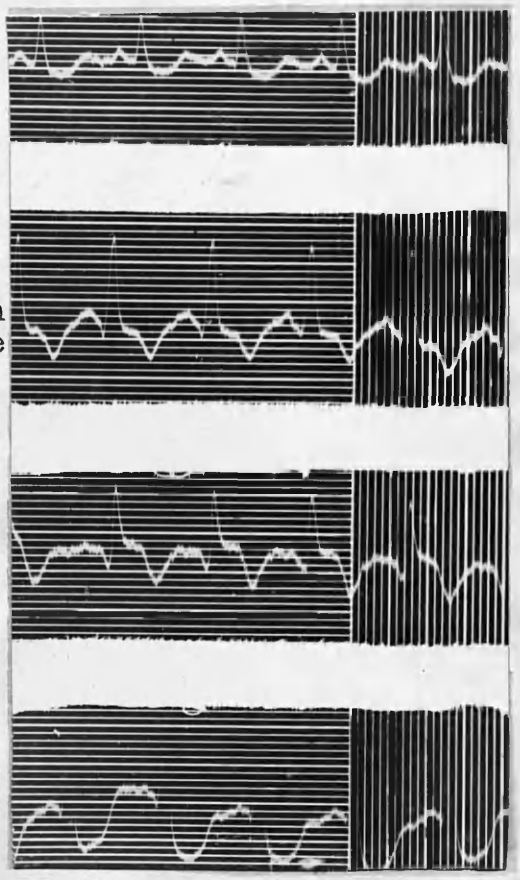
Healing stages of myocardial infarction.

Following myocardial infarction the electrocardiogram undergoes a characteristic evolution usually returning towards the normal. This process involves the gradual return of the ST segment to the isoelectric level and the T waves to their normal size and position. This process of evolution may become arrested at various stages in its progress, (usually the later stage) and a condition depicting chronic or protracted coronary insufficiency may remain. This arrest usually takes place at the coronary T wave stages but it is not usual for the ST

segment to remain abnormally elevated or depressed beyond a certain period of time, but in certain cases this too can occur.

The following case is an example of coronary T wave changes in a healing posterior infarction.

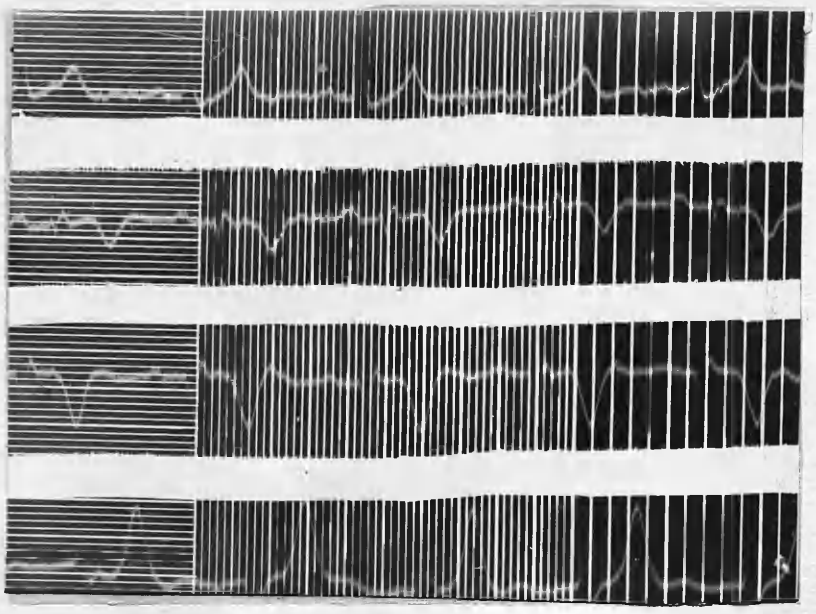
Mr.N. Aged 55 Seen complaining of precordial pain on exertion of few days duration and with pain on left side of neck for about 14 days before this. This patient was sent home to rest and the following day developed intense substernal pain with no radiation. B.P. 120/90 and on auscultation no abnormal cardiac sounds were present but extensive crepitations were audible over the lung tissue along the left cardiac border. Considerable tachycardia was present Rate 120 and this persisted for about 14 days At this time the following ECG was taken and shewed the characteristic changes of a posterior infarction with depression of ST1 and elevation of ST 2 and St 3 with deep inversion of T2 and T3 of the coronary type. The chest lead shewed depression of ST segment and T wave.



Such T wave changes seldom remain as a permanent feature and no doubt this record will undergo further changes towards normal before any residual T wave could be regarded as a permanent indication of protracted coronary insufficiency.

These T waves usually shew the characteristic contour of the coronary T wave often remaining for a considerable period up to a year or more following the infarction, but in most cases the tendency is for a return to normal T waves within a period of months. The anterior infarction tends to leave an inversion of T1 and the posterior infarct an inversion of T3 and T4; with the ST segment more or less at normal level. On the other hand a very large T wave is perhaps one of the most useful indications of protracted coronary insufficiency, with perhaps the exception of definite ST changes.

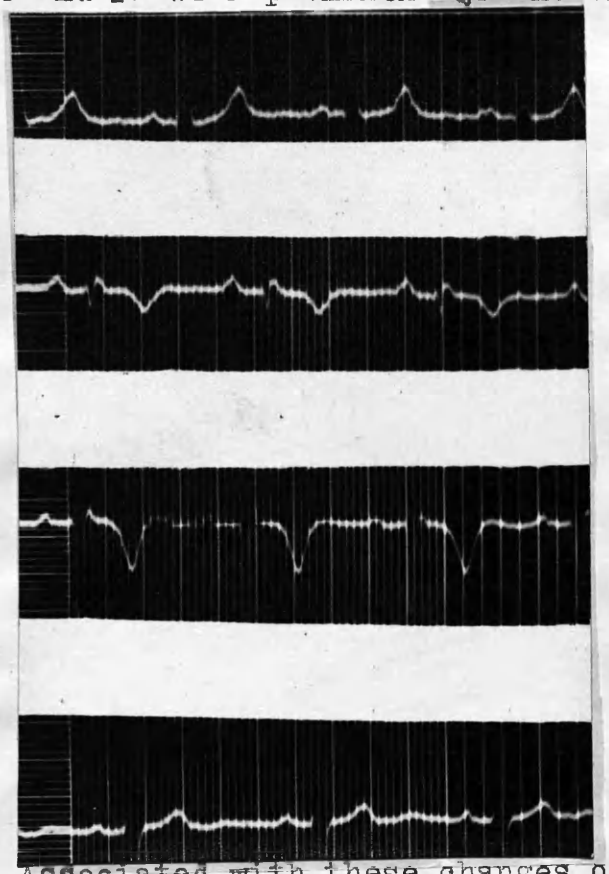
This record is taken three and a half months after a posterior infarction and at this time the patient was complaining of substernal discomfort on exertion, but diminishing in intensity.



Here is a typical example of the high T wave in the

chest lead of coronary type, with definite coronary T waves in lead 11 and 111, with prominent Q3 and small Q2.

In this particular case a further record taken one year afterwards shews later evolution of T wave changes which were found to be unchanged at the end of another twelve months. Here the changes obviously have become stabilised with a now normal chest lead and only inversion of T3 and T2 with prominent Q3 and small Q2 remain.



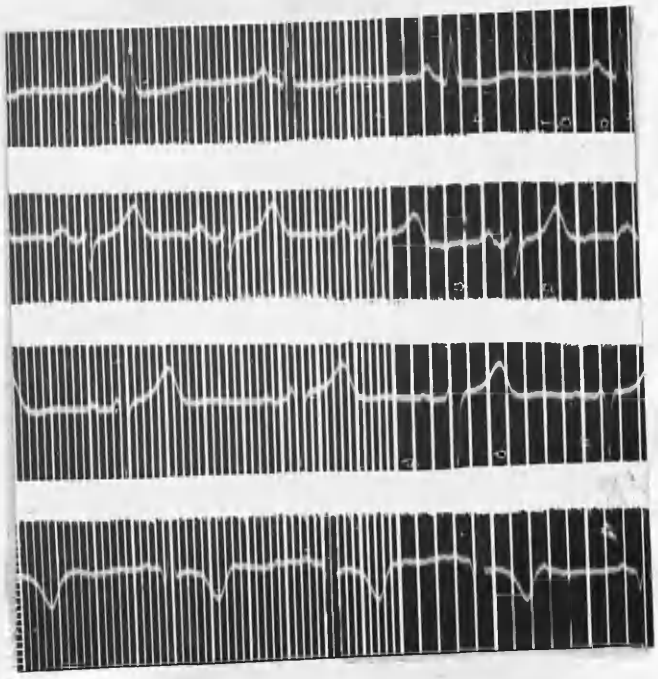
Associated with these changes of chronic coronary insuffieiency we usually find typical Q waves in leads 1 or 11 or 111 and absent R wave in the chest leads. Such T wave changes are especially significant in this investigation

since they are often the only indication of coronary disease. The description of this stage as protracted coronary insufficiency by certain writers seems very apt and these various stages in the evolution of the electrocardiogram are important. The early T and ST changes correspond to the late changes of the infarction, and finally the T wave is seen to undergo a process of increase and decrease (the so called waxing and waning of the T wave) before it reaches a stable stage.

The rate of ST-T evolution from abnormal to normal gives us some indication of the rate of the healing process and likewise the waxing and waning of the T wave is some indication of the stage which the healing process has reached. Any sudden interruption of this normal process of evolution of the T wave particularly if associated with ST changes may be an indication of a further myocardial infarct. In some cases the T wave may not reach great voltage before the onset of the waning stage, but finally a stage is reached when the T wave becomes the dominant feature of the electrocardiogram. This tendency on the part of the electrocardiogram to return to normal is usually a dominant feature but is not invariably the case, nor is it by any means certain that such a return to normal is "per se" an advantageous factor. It seemed however in this series that cases shewing such coronary T wave changes as a lasting feature were more prone to anginal

pain, but this appears to be a matter still under discussion. One important error can occur in mistaking the early evolution of an acute myocardial infarction for a chronic coronary insufficiency, since these two conditions can sometimes give similar records. It is important therefore to follow up the record in any case of doubt with a record taken at an interval of a few days.

In this group of changes associated with chronic or protracted coronary insufficiency we find occasionally, such QRS changes as ventricular preponderance heart blocks and various arrythmias. It must again be remembered that coronary sclerosis is not alone in producing a limited coronary supply and that such conditions as pericarditis, pulmonary embolism, acute nephritis, severe anaemias dissecting aneurism and certain drugs can produce similar changes. In this group of chronic coronary insufficiency the more characteristic changes such as coronary T wave Q waves and low T wave occur frequently. A low T wave particularly occurring in Lead 1 or 11 can often be a transition stage between an inverted and an upright T and is always regarded with great suspicion. This record is an example of chronic coronary insufficiency in a man aged 53 years.



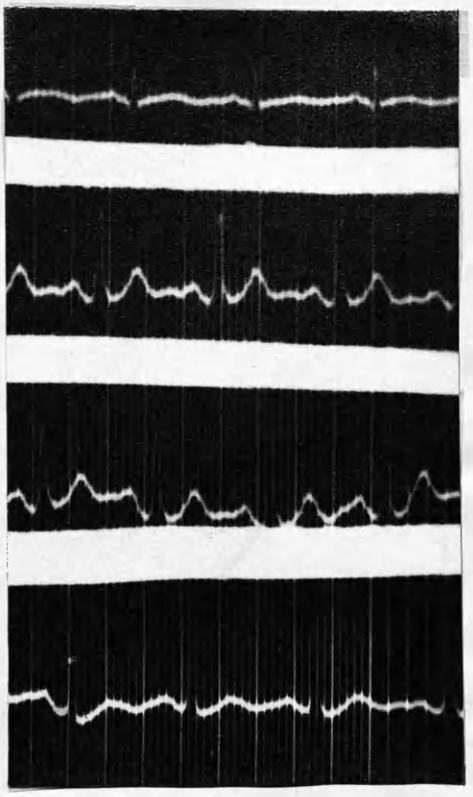
Complaining of pain across chest on exertion, such as walking, of three months duration. This patient gave a history of a severe attack of pain while returning from work which persisted the whole day and which was the forerunner of his present substernal discomfort.

When this record was taken his B.P. was 170/90

The only abnormality on auscultation was the accentuated second aortic sound.

ECG here shews coronary T wave in lead IV with small Q4 and Q3

Here an isoelectric T 1 with coronary type T waves in all other leads gives a record of healing or healed infarct of the posterior type. A further record taken some months later shewed very little change and one is justified in assuming that some degree of permanent coronary insufficiency exists in this case although no doubt this record may shew a greater return to normal. On the other hand the patient's story indicates a waning exercise tolerance, which is more suggestive of a chronic state. The following case might illustrate this.



Man aged 43 complaining of a pain in his chest with substernal location, occurring on cycling to work. This pain had continued for some time, sometimes occurring in bed, on one occasion lasting most of the night.

He was confined to bed by his Doctor for five weeks and since then now has pain on walking for 8 - 10 minutes only, when he has to stop and rest.

When seen at the time this record was taken his B.P. was 135/90, an accentuated first mitral sound being the only abnormality.

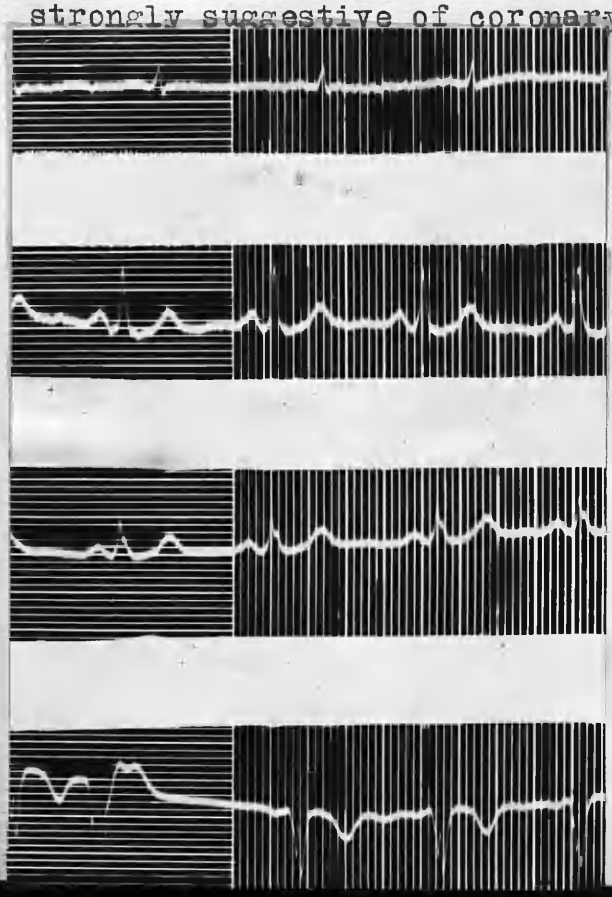
On screening he appeared to have some enlargement of left ventricle and low diaphragm.

This type of case can be exceedingly difficult and the severe attack of pain only seven weeks ago does not appear to have been due to an actual infarction but the low T1 in this record is certainly suggestive of some chronic coronary insufficiency.

Other changes associated with coronary insufficiency.

Notching and Slurring.

Notching or slurring is a frequent occurrence in coronary sclerosis and is of diagnostic importance if sufficiently pronounced. It is important to remember that in dealing with QRS changes we are considering changes indicative of a more permanent damage to heart muscle, since any changes here are due to altered pathways of the electrical impulse in the cardiac muscle. Such alterations appear as a slur or notch of the QRS or widening of the QRS wave indicative of delayed interventricular conduction. Notching of the QRS is of significance if high up near the peak of QRS and is still more significant if associated with T wave changes in which case it is strongly suggestive of coronary disease.



Mr. G. Aged 55
This is a record from a patient with a definite myocardial infarction two years ago. He now complains of some substernal pain on exertion.

When seen his B.P. was 170/100

The ECG taken at the time shewed some notching of QRS in lead 111 and definite notching in lead IV associated with coronary T wave, and some degree of intraventricular block with QRS of .10 sec.

Lead 1 shews an isoelectric T wave and all these findings confirm the diagnosis of chronic coronary insufficiency although such slurring and notching of the chest lead is not always regarded as being abnormal, and notching can occur as a normal variant in lead 111 where the QRS is small and where R1 is approximately equal to R2.

It is interesting to note that in the same record notching occurs in the same position of time in each QRS and is sometimes manifested as a notch in one lead or a slur in the other.

The increased duration of QRS.

Cases shewing varying degrees of widening of QRS wave beyond .12 of a second occur not infrequently in coronary insufficiency and are of considerable clinical significance. Many cases giving a clinical history of pain on exertion are found to shew varying degrees of axis deviation, usually the left, associated with hypertension or aortic disease and intraventricular block.

The Hypertensive Electrocardiogram.

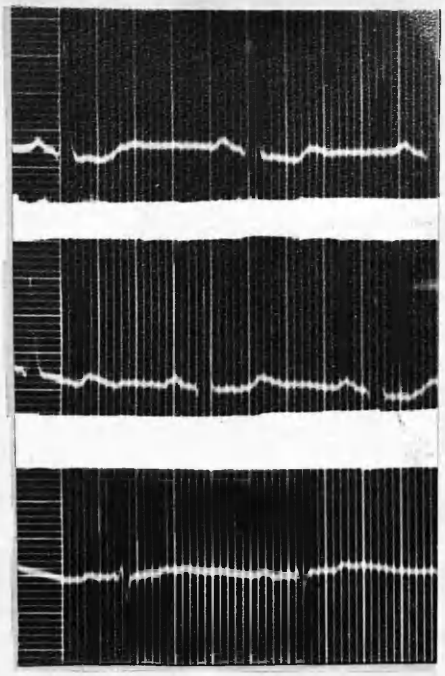
The existence of hypertension produces definite progressive changes in the electrocardiogram, all of which can occur in association with coronary insufficiency.

In some cases hypertension can shew no abnormalities in the electrocardiogram, and as a general rule pain was not a dominant feature of this type. As hypertrophy of the ventricle develops a definite left axis deviation appears in the electrocardiogram with gradually increasing R1 and S3. A later stage of left ventricular preponderance is associated with large left ventricle and depression of the ST segment in lead 1 and then in lead 1 and 11. The ST segment is deviated in the same direction as the T wave in contradistinction to the coronary curve where the ST segment is opposite to the T wave. In the more advanced stage some degree of QRS slurring and notching with intraventricular block occurs, and finally even definite coronary T wave changes suggestive of myocardial injury can appear. These ST-T changes are grouped by some writers under the term ventricular strain although there seems to be some objection to this term. These changes occur more readily in left axis deviation than in the right, since the right ventricle is not capable of hypertrophy to the same extent as the left. The developing intraventricular block is here probably due to hypertrophy and not the result of a conduction defect. Some authorities maintain that the T wave inversion is in the nature of a relative coronary insufficiency (Katz) owing to an increase of ventricular muscle beyond the

capacity of the coronary vessels to supply it. Pardee (73) p.98 discussing ventricular hypertrophy maintains that a widening of the QRS in advanced left ventricular preponderance can be as great as .12 of a second without any actual defect in conduction, but this whole question of whether a left ventricular hypertrophy can, if long continued, produce the condition of left bundle branch block seems to be still a matter for debate. For our purpose in the investigation of these cases there seems little doubt that where these changes occur supported by a suggestive clinical history the outlook is relatively poor, and statistics shew a comparatively high mortality rate where such T wave changes associated with hypertension and wide QRS are found. (Dressler (67) p463)

Masters (47) Quotes several examples of the evolution of typical bundle branch pattern from left axis deviation over a period of years, yet on the other hand, many cases of extreme left ventricular enlargement can shew a normal QRS duration, and in fact many hypertensive cases can shew a normal electrocardiogram as stated above. The following record is taken from a case showing developing hypentensive electrocardiogram.

Man aged 44 Complaining of headaches, giddiness etc. with some precordial discomfort.



He was found to be hypertensive B.P. 220/100

No renal changes or retinal changes.

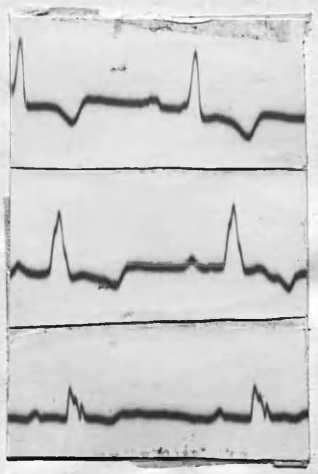
On auscultation an aortic 2nd sound was found to be accentuated.

X Ray findings shewed typical aortic configuration with large left ventricle.

ECH shews QRS of normal duration with depressed ST 1 diphasic T1 with pronounced left axis deviation and high voltage QRS

It is interesting to note how often an accentuated second aortic sound can continue after a fall in blood pressure. In this case he was considerably improved clinically by a lumbar sympathectomy with B.P. 150/90 three months afterwards. Here the accentuated aortic sound with large left ventricle were still obvious after treatment.

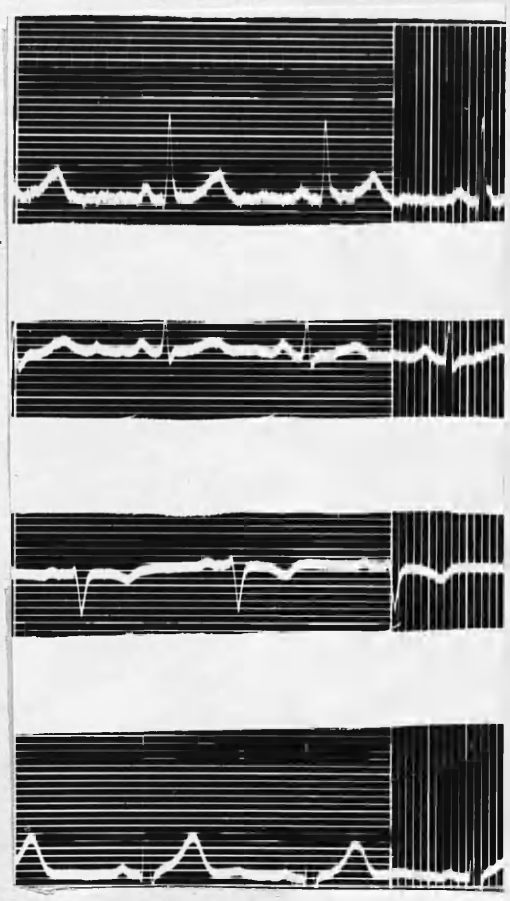
The advanced form of wide QRS with inversion of T1 and T2 with no axis deviation seen in the following case:-



This record was taken in the case of a man aged 42 years with advanced aortic regurgitation and general cardiac enlargement. He was subject to considerable anginal pain with considerable dyspnoea of six years duration. A high degree of hypertension existed 270/70 the wide pulse pressure due to the aortic regurgitation. Considerable slurring QRS complexes with great degree of widening suggests considerable myocardial damage and some delay in AV conduction.

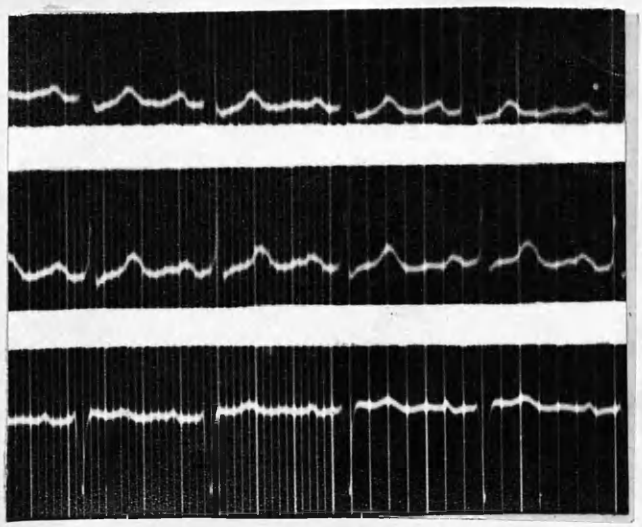
PR interval .32 seconds. The absence of pronounced left axis deviation may be due in this case to the effect of both right and left ventricular enlargement.

Hypertension can be similated by a high position of the diaphragm producing an upward displacement of the heart and giving a record of left axis deviation on the electrocardiograph. The following case is taken from a patient aged 52 years - a Free French Air Pilot who had until a few weeks previously been flying at high altitudes. He was seen in Hospital complaining of left submammary pain occurring mostly in bed and often peristing throughout whole night. He described the sensation of a flow of blood inside the left arm in association with the pain. He was of a highly strung disposition and gave a history of nervous breakdown in 1936 with similar symptoms. On examination no abnormality was found on auscultation. B.P. was found to be 120/80 ECG shewed no abnormality other than left axis deviation with inversion of T3. The asymmetrical type of T3 inversion was noted and on screening this patient shewed a high diaphragm with wide chest. Subsequent ECG records taken shewed no change in this contour and it was assumed that the changes were due to transverse heart.



It is usual for such records to shew an inversion of P3 as well as T3 changes and left axis deviation where the abnormal position of the heart is a causative factor.

It is possible of course for hypertension to exist with considerable clinical manifestations without pronounced electrocardiographic changes.



This is a case of hypertension in a woman aged 45, of some years standing.

B.P. 210/100.

This patient now gets persistent ache under sternum on exertion, also at rest.

On screening typical aortic configuration of the heart was present.

The ECG shews considerable degree of left axis deviation with early changes chiefly depression of RT1 and RT2 with corresponding elevation in RT3 but so far no T wave changes.

Such changes are frequent in this series and are of considerable value in assessing the degree of cardiac involvement in a suspicious clinical history.

In right axis deviation the wide QRS is not common and this condition is not of such great diagnostic importance since it is not found frequently in association with cardiac pain, except perhaps in cases of mitral regurgitation following enlargement of left heart before

involvement of right. Right axis deviation is more usually found associated with conditions involving strain of the right ventricle, such as mitral stenosis, cor-pulmonale, embolus of the pulmonary artery and in these cases dyspnoea is the more predominant symptom.
Where chest pain does occur the appearance of right axis deviation in the electrocardiogram calls for an investigation into the function of the lesser circulation since not infrequently such conditions as pulmonary embolism can produce a chest pain and sudden appearance of a right axis deviation although not invariably so. Sometimes a large S1 can appear with this condition without right axis deviation, and not infrequently a Q wave develops in Lead 111. The recognition of these patterns of advanced left and right ventricular preponderance and their differentiation from bundle branch block is important here since these conditions are all frequently occurring in association with cardiac pain of coronary insufficiency.
In advanced left ventricular hypertrophy the ST segment and RT junction are usually entirely below the electric line in lead 1 and above it in lead 111. In lead 111 the ST segment is concave upward and the associated QRS and T waves are usually of large voltage. The T wave also does not as a rule conform to the coronary T wave.
In right axis deviation an inversion of T3 occasionally occurs then follows the inversion of T3 and T2, usually

with a normal QRS.

The causes of left ventricular hypertrophy include aortic regurgitation and stenosis, hypertension coronary arterio sclerosis and occasionally mitral regurgitation. This particular group is more frequently associated with clinical symptoms of coronary insufficiency such as cardiac pain. Right ventricular hypertrophy is usually associated with pulmonary disease, emphysema, tuberculosis, certain congenital abnormalities, pulmonary stenosis, septal defects, patent ductus arteriosis, mitral stenosis and mitral regurgitation. The latter only produces right ventricular hypertrophy through dilating the left ventricle so that the early signs of mitral regurgitation are usually left ventricular hypertrophy and cardiac pain is not a frequent accompaniment of this group.

Importance of Q and T wave changes.

Many cases of coronary sclerosis shew large Q waves in Lead lll but this Q wave can only be accepted as significant if it conforms to certain accepted limits. It seems to be recognised that a Q3, to be of diagnostic value, should be at least 25% of the largest R in any limb lead. If right axis deviation is present or if the R wave is followed by an S wave, or if the QRS is of the M type, then the Q3 has not the same value.

Likewise a Q2 of three millimetres or more or even a small Q1 can be regarded as abnormal especially in a right axis deviation. The larger the Q3 in relation to the size of the QRS the greater is the chance of coronary sclerosis but it seems doubtful if we should include those QRS waves with an absent R wave (the QS waves) in this group. When an abnormal Q3 is associated with a T3 or T2 inversion then the chances of an underlying coronary sclerosis is much greater. The large Q3 is a frequent finding in patients with the anginal syndrome but it should be remembered that it can occur in rheumatic hearts, pericarditis of rheumatic origin, and pulmonary embolism.

Where a Q wave varies with respiration sometimes disappearing this does not seem to affect its diagnostic importance.

Duration of Q3.

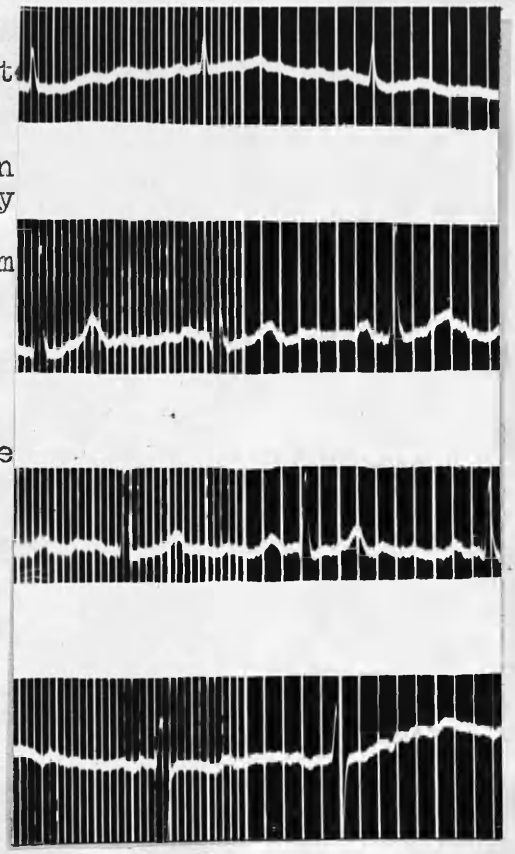
It is maintained by some writers that where the duration of a Q3 exceeds .04 of a second or more coronary sclerosis may be assumed to be present, particularly if the Q3 is associated with a Q2.

T wave voltage.

Any condition producing a depressed state of the myocardium of which coronary disease is one can induce lowered T wave voltages, but this can be produced by

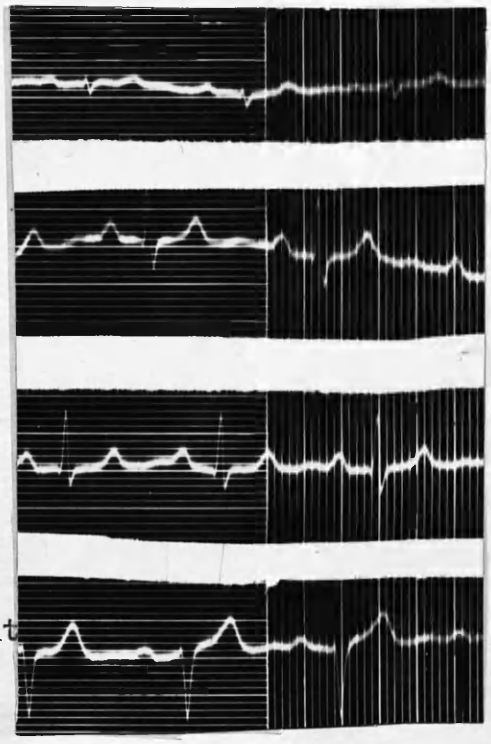
rheumatic fevers, chronic valvular disease or even failure. If low voltage T is found with notched QRS it is usually the result of diffuse disease of the myocardium. It is important here to differentiate between low amplitude (in one lead) and low voltage (in all leads). Low amplitude of the T wave in one lead is frequently a transition stage in the evolution of an inverted or abnormal T wave and as such is of great diagnotic importance particularly if occurring in Leads 1 and 11 or in the chest leads. A low T1 is a common finding in patients with the anginal syndrome in thie series, and sometimes this remains as a permanent feature in successive records, thereby suggesting that in itself it is an indication of chronic coronary insufficiency.

The following cases can illustrat



Mr.G. Aged 57 years. Rate 80 B.P. Seen complaining of pain across chest but predominantly substernal with radiation along ulnar aspect of left arm to middle fingers, of three months duration. This occurs now on walking a distance of 100 yards and disappears with a few minutes rest. The onset here appears to have been gradual and no dramatic incident suggestive of infarction can be ascertained. This patient gave a history 12 months ago of occasional tightness of chest on exertion and ECG taken then shewed no essential difference from the present. Here undoubtedly is an indication of a chronic coronary insufficiency, the record shewing an isoelectric or low T1 with diphasic T4 as the only abnormality.

In some cases a low T1 or absent R in the chest lead may be the only indication of coronary disease. The following case was seen with a history of recurrent attacks of pleurisy along the pleuro pericardial border. Here the onset is usually dramatic with left sub-mammary pain radiating down left arm (ulnar distribution) into left axilla. The patient is frequently seized while at work.



On examination a pleuro pericardial friction rub appeared lasting about two days. Because of the peculiar radiation of the pain an electrocardiogram was taken four weeks after a recent attack. The record shewed absent R wave in the chest lead from the apex with low T1, high peaked P wave in lead 11 and lead 111 with some degree of AV block PR interval .24 of a second. Here the tendency to right axis deviation with high P wave suggests a mitral stenosis and this was supported by screening.

Auscultation shewed accentuation of first mitral sound The record also supports a history of some old myocardial infarction.

BUNDLE BRANCH BLOCK.

The occurrence of certain abnormalities of the T wave and QRS together constitute a group of electro-cardiagraphic changes found with considerable frequency in this series, and is of supreme diagnostic importance.

it is only possible to diagnose this condition by means of the electrocardiogram and Cowan (1930) is quoted by Bramwell (68) as stating "You must use wet fly if you want to catch the bundle branch block". This finding is of great significance particularly in cases of anginal pain since it considerably alters the prognosis.

The typical bundle branch pattern consists

(1) of abnormally wide QRS with some slurring or notching

(2) the largest QRS is usually oppositely directed in leads 1 and 111 i.e. the discordant type, but others have the QRS 1 and 111 in the same direction i.e. the concordant type

(3) the PR interval is usually normal but a benign type with short PR interval described by Hunter Papp & Parkinson (48) exists but varying degrees of AV block are found in this condition.

It is now regarded that this condition is due to some defect in the conduction system compelling the impulse to spread slowly through the muscle of the septum so that it does not reach both ventricles simultaneously. From the chief deflection in the leads it is possible to determine which ventricle is activated first. A recent method of differentiating Bundle Branch Block from the advanced form of ventricular hypertrophy is described by Pardee (69) and consistsin the calculation of

the delay in the occurrence of the peak of the R wave from the commencement of the QRS in the chest leads taken from both sides of the sternum and the apex. Pardee maintains that a degree of delay of the R wave over the right ventricle (right of the sternum) is attributed to right bundle branch and conversely in left bundle branch block the peak of R is much earlier over the sternum and to the left of the sternum than over the apex and beyond.

This delay in the occurrence of the peak of the R can be as much as .08 of a second after the commencement of the QRS in bundle branch block but never occurs to this degree in other forms of intraventricular block such as advanced left ventricular preponderance although these may show as much as .06 of a second.

Left Bundle Branch Block is found in two types

Type 1 The discordant type Lead 1 Chief deflection is R though a small Q may appear and T wave is usually down Lead 111 chief deflection is S though there may be a smaller R, the T is upward.

Type 2 The concordant type

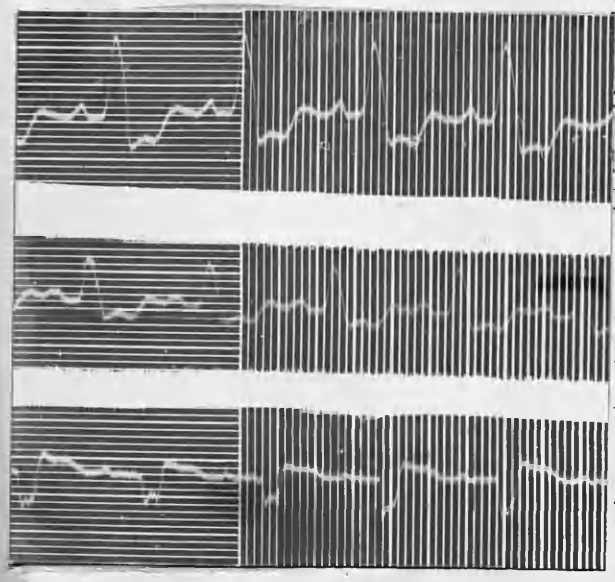
Lead 1 is the same as for discordant type

Lead 111 chief deflection is R and there is a small S. T usually upwards.

A considerable degree of notching occurs in the QRS and this seems to be a useful guide to the degree of myocardial involvement. In this series where a great

degree of widening or notching of the QRS was found, the general progress of the patient seems to have been more rapidly downwards, and where bundle branch block appears without much widening of the QRS the history seems to have been more benign. It is a singular fact that in investigating cases with the anginal syndrome left bundle branch block was the usual finding, and in view of the frequency of conditions producing left ventricular preponderance such as hypertension and aortic disease with clinical symptoms of coronary insufficiency there would appear to be some association between left ventricular hypertrophy and the occurrence of bundle branch block.

The following cases can illustrate the type of record obtained in quite a few patients with coronary insufficiency:-



Mrs P. Aged 72 years. Rate 96 Became conscious of heart six months ago, had to keep stopping for breath began experience nocturnal dyspnoea.

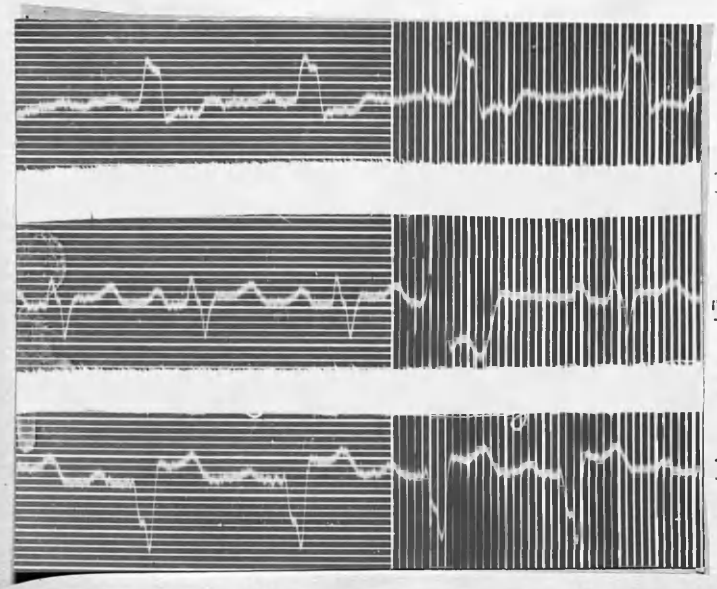
Three months ago began to get pain across chest and lower end of sternum on effort.

On examination rough aortic systolic murmur conducted to neck vessels.

B.P. 110/78 Apex beat displaced downwards to left.

ECG here shews left bundle branch block discordant type Type I QRS duration .16 of a second.

The degree of dyspnoea is no doubt indicative of general enlargement of the heart with a high degree of myocardial damage, and no doubt her aortic stenosis is a factor in her coronary insufficiency.



Mrs J. Aged 58. Complains of substernal pain radiating under left breast to back and down posterior aspect of left arm to elbow.

Pain induced by quiet walking or emotional upset such as air raid etc.

This patient dates her history from fall on chest two years ago when ribs were fractured.

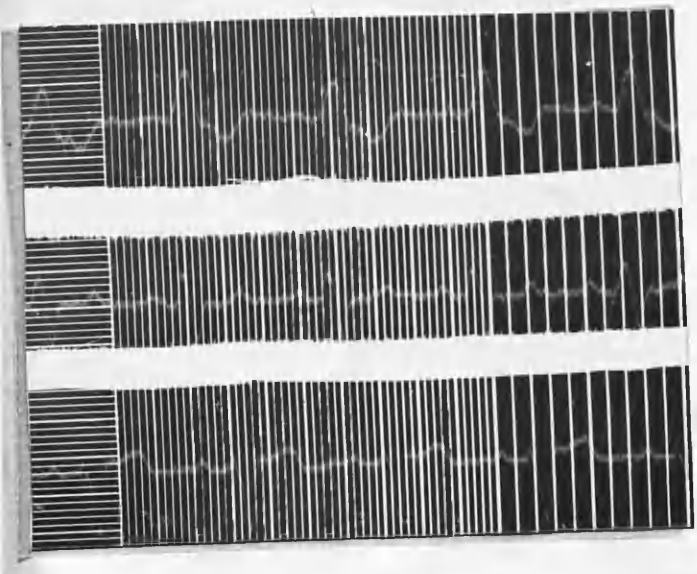
ECG shews left bundle branch block with wide and notched QRS and ventricular extra systole in lead 11 Type I.

Auscultation no abnormality.

B.P. 150/90

This case raises the interesting question of the possibility of cardiac trauma in the production of myocardial damage or damage to bundle branches.

The following case is a record from a patient with more advanced anginal symptoms and here of late dyspnoea has become the predominating feature.



Mr. M. Aged 70. Complains pain on exertion 12 months duration now occurring with gentle walking few yards.

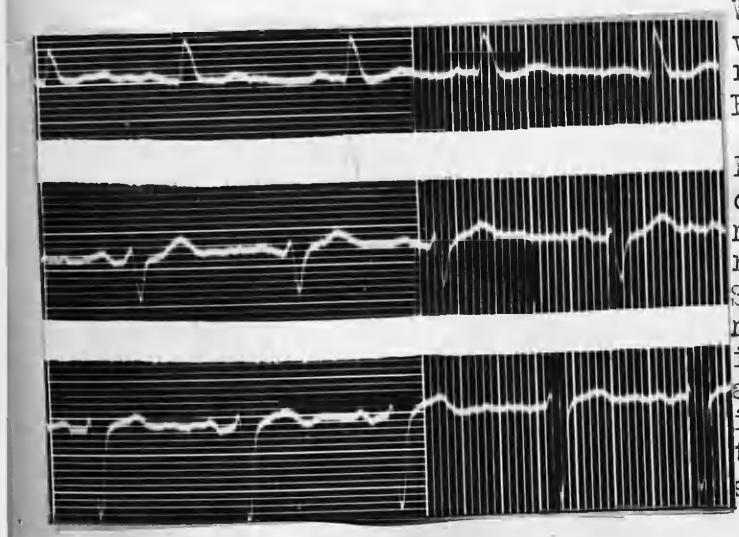
Pain is epigastric in type radiating along medial aspect of both arms to elbow.

Auscultation No significant abnormality.

B.P. 190/100.

ECG shews left bundle branch block with wide QRS .20 of a second. Type I.

The following record is of a slightly different type since here the QRS is .12 of a second and no significant notching or slurring occurs.



Mr.E. Aged 72. Gives a history of attack of chest pain 2 years ago with some slight discomfort under sternum on hurrying only. Whether his earlier attack was a coronary thrombosis is now difficult to make out.
B.P. 180/100

Interesting feature of this case is the presence of intermittent claudication more pronounced in left leg. Similar symptoms of intermittent claudication appear in the case of his daughter, and probably his coronary insufficiency might be in the nature of a vasomotor spasm.

ECG shews left bundle branch block of the usual type with low T1 a significant finding. Type II.

It seems to be generally accepted that in complete bundle branch block the QRS should be greater than .12 of a second. Many cases of left ventricular preponderance shew a QRS as wide as .12 but not more and should be regarded as incomplete bundle branch block. In so called arborisation block which is supposed to be due to damage to the sub-endocardial ramifications of the branch the QRS complex is usually of small voltage and is wide with considerable notching. This type is found frequently in myocardial disease but is not frequently associated with cardiac pain.

Various degrees of incomplete block can be found and a pattern with a wide S wave is supposed to have a more hopeful outlook, and is occasionally found in normal hearts.

Right Bundle Branch Block.

This condition is found rarely and was not found in any case in this series of cardiac pain.

Bayley has described four types and Wilson has described a fifth. This can be tabulated as follows:-

Type 1	Lead 1	Chief deflection broad S with small R1, T is upward.
	Lead 111	Chief deflection R which is broad and notched with downward T
Type 11	Lead 1	Broad S but R larger than S T is upward
	Lead 111	Chief deflection is R which is broad and notched with T downward.

Type III Lead I Large R. broad S.

Lead III Slender downward spike of some size preceded by small upward deflection and always followed by broad summit T may be downward or upward.

Type IV Lead I Chief deflection is R followed by broad smaller S T upward or downward.

Lead III Small R followed by broad inverted spike. T upward or downward

(Wilson) Type V Lead I R and S both of small amplitude T upward or isoelectric

Lead III Broad S with upward T

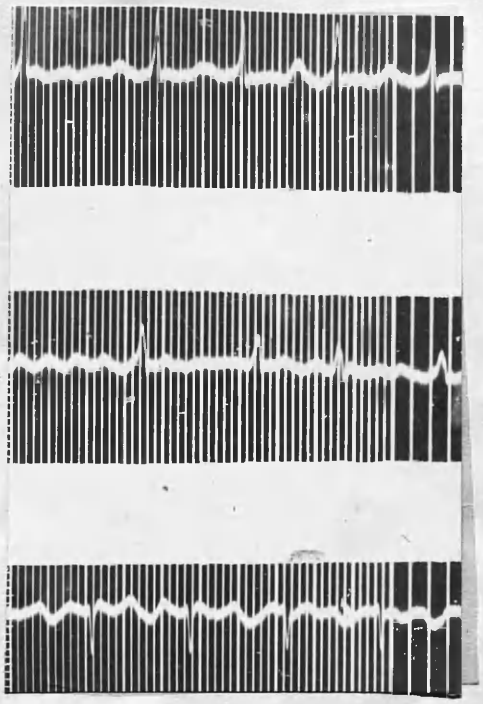
There seems to be no special clinical significance attached to any particular type of bundle branch block although the left type appears to be the more usual finding in cases with anginal pain. This is no doubt due to the fact that the conditions producing left axis deviation such as hypertension, or aortic disease, are more frequently found in association with cardiac pain. Right bundle branch block seems more frequently associated with conditions producing right ventricular strain such as pulmonary conditions, mitral stenosis already enumerated. There seems little doubt that the finding of bundle branch block diminished considerably the prognosis although rare cases have been known to continue for many years with this condition, but generally speaking cases with

anginal pain shewing bundle branch block do badly. From this series it appears that those cases shewing wide and notched QRS have a more rapid deterioration of their clinical condition and are usually associated with symptoms of coronary insufficiency and dyspnoea.

Disorders of Rhythm.

Certain disorders of rhythm are frequently associated with pain of the anginal type, such as paroxysmal tachycardia, simple tachycardia with high ventricular rate, and even rare cases of bradycardia.

An attack of paroxysmal auricular flutter fibrillation can produce substernal pain although it is an unusual association.



Mr B. Aged 60.
This patient gets attacks of rapid beating of the heart during any emotional upsets such as air raids etc. This is associated with substernal pain of varying degree of intensity and which passes off with return to normal rhythm.

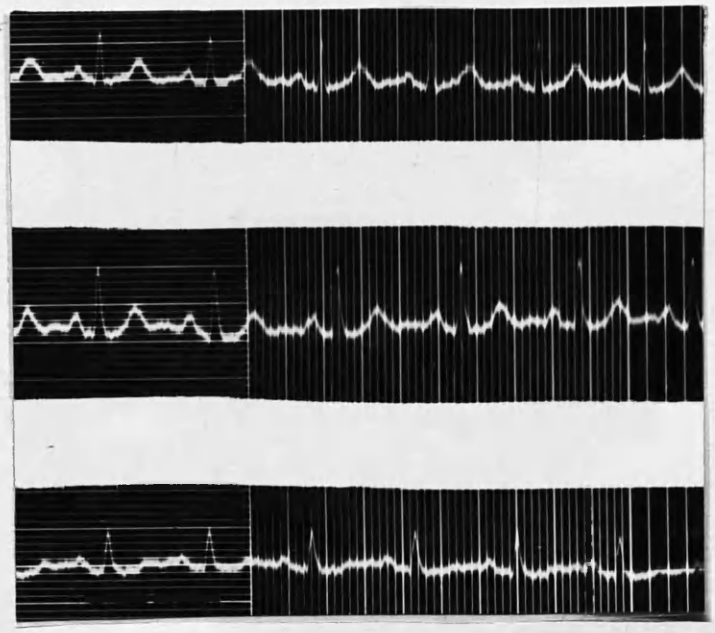
This record was taken during such an attack and shews coarse fibrillation with complete irregularity of rhythm.

The record taken at normal times shewed sinus rhythm with an occasional completely dropped beat.

It is an unusual occurrence to find anginal pain associated with auricular fibrillation and where fibrillation is persistent the pain usually disappears. As a rule such conditions suggest involvement of the auricle as the dominant feature in the coronary disease and pain therefore is not prominent.

Simple tachycardia if associated with a high ventricular rate can often produce substernal pain particularly if associated with hyperthyroidism.

The following case is an example:-



Mrs A. Aged 40. Complains of substernal pain on any exertion often when sitting still. This has persisted for 5 months with intermissions.

There is a neurasthenic background here and she gives a history of nervous breakdown some years ago.

B.P. 120/80

ECG shews simple tachycardia

Rate 120 regular rhythm

Bradycardia.

It is not usual for cases of simple bradycardia to experience substernal pain since it is generally recognised that any deficiency in coronary circulation is compensated for by greater systolic impulse, in fact such cases often shew pulse volume 50% greater than normal, and the actual excursion noted in systole on/

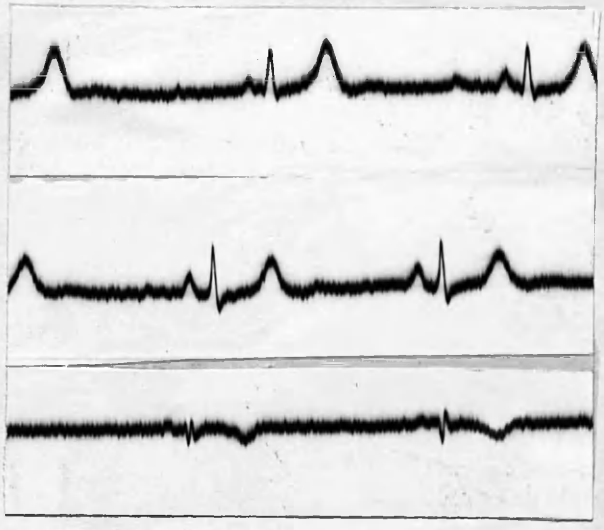
fluoroscopy can be as much as 1.5 or more centimetres. (70) (Roesler)

The following however is such a case

Mrs L. Aged 47. Complains of lack of energy with great dyspnoea. Nervous breakdown two years ago. Complains of left submammary pain radiating into back and ulner aspect of left arm. This occurs on moderate exercise and sometimes in bed.

Examination B.P. 160/80. Rate 44.

On screening systolic diastolic excursion at least 1 centimetre with considerable enlargement of the heart - a usual finding in bradycardia.

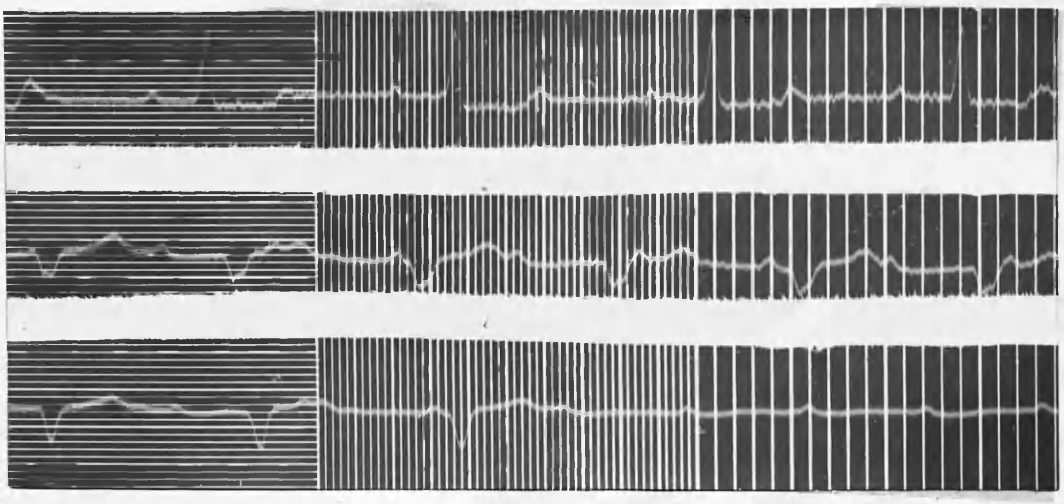


Other types of arrythmia can result from coronary disease. This can include multiple ectopic beats, varying degrees of heart block, auricular fibrillation and flutter following in a case of anginal pain.

In some cases complete heart block can be present for some years and the only indication may be a slow regular rhythm. In these cases the nutrition of the centre controlling the idioventricular rate can be altered by impairment of the coronary circulation sometimes of a temporary nature. In such cases the regular ventricular

contraction becomes irregular and often stops for varying periods at a time - the so called Stokes Adams attacks.

The following case is a record from a patient with such a condition:-



Mrs W. Aged 60 Seen in Hospital with attacks of unconsciousness occurring frequently and varying in intensity She gave a history of substernal pain on exertion with considerable dyspnoea B.P. 120/100 Rate 40 with periods of absent pulse associated with her attacks of unconsciousness; these lasting 60 seconds or more. ECG here shewed left bundle branch block with complete AV block although Lead 1 suggests a 2 - 1 heart block, but Lead 11 shewed regular auricular rate of 120 with ventricular rate of 60 but no evidence of conducted auricular impulse. Lead 111 shewed complete absence of ventricular contractions and this persisted for sixty seconds, when the patient recovered consciousness.

This condition was probably some impairment of coronary circulation with diminished nutrition of

conduction system. The patient recovered and was discharged in a few days.

Exercise and Low Oxygen Tests.

Attempts have been made recently to induce myocardial ischaemia by low oxygen administration or exercise in an attempt to find some definite cardiograph changes which could be construed as proof of the existence of coronary disease.

Experiments conducted by Rothschild & Kissin by administration of low oxygen mistures showed that both pain and cardiograph changes could be produced. Similar experiments have been conducted by Barrach, Steiner[51] et alia where low oxygen mixtures 10% (with 90% nitrogen) were administered in known cases of coronary sclerosis and here similar results were obtained. Wilson & Johnson[52] conducted similar experiments and emphasised the increase of S deflections in lead 11 and 111 with pronounced downward deflections in these leads.

One important finding was that the changes were similar in magnitude to the changes following occlusion but that where the changes in occlusion were discordant those of coronary insufficiency were concordant.

Levy[53] has investigated this very carefully and found definite changes in patients with diminished coronary reserve not present in the normal His conclusions are that, where the pain appears during the first 10 minutes of anoxemia coronary disease is very likely. His electrocardiograph findings in 1100 cases were as follows:-

1. Arithmetic sum of RST deviations in all four leads 3 m.m. or more

2. Partial or complete reversion of T wave in lead 1 accompanied by RST deviations of 1 m.m. or more

3. Complete reversal of the T wave in lead IV regardless of RST deviation

4. Partial reversal of direction of T wave in lead IV F accompanied by RST deviation of 1 m.m. or more

In some cases reciprocal RST changes do occur and it is suggested by some American writers where this is so we may be dealing with a patient who has previously had a coronary occlusion. The important thing from the diagnostic point of view is the rapid return to normal on the cessation of the induced anoxemia. Barnes[54] quotes the findings of Wood & Co. in their investigation, where significant changes were found in only 50% of patients during an attack of anginal pain and their cardiograph findings were as follows:-

1. Altered ST segment of T wave.

2. Evidence of conduction defects in AV bundle or bundle branches.

Sherf & Boyd (55) lay great stress on the type of segment change rather than the amount. Masters (56) likewise stresses the RST depression as an indication of coronary insufficiency including with it T wave inversion. He puts forward an interesting suggestion that in the infarction the damage extends throughout the whole cardiac muscle from endocardium to pericardium and implies that elevation of the RST segment is associated with epicardial damage only and depression with sub-endocardial damage.

There seems to be little doubt from the study of the findings of these various workers that definite and concrete changes do occur in coronary insufficiency with this test but only in a limited number of cases.

EXERCISE TEST.

A similar method of inducing relative coronary insufficiency by means of exercise has been adopted and seems to me to have the greater advantage and simplicity of application. Sherf & Boyd (55) in discussing the application of this test point out that 60% of cases of anginal pain show normal electrocardiographs. They also demonstrate that T and ST changes can occur in the untrained person after strenuous exercise, but not in the normal individual on mild exertion. In patients with coronary sclerosis on mild exertion they found marked

acceleration of heart rate and depression of ST 2 and 3 with even Q wave changes for a short time (10 - 30 minutes) after exercise.

As the result of these findings by various workers it can now be assumed that the changes in anginal cases on exercise resemble changes occurring in a spontaneous attack of paroxysmal pain. They regard as a positive test depression of ST segment in Lead ll and lll and lowering of the T wave.

Twiss & Socklow (57) describe their results of this test in patients with and without angina and came to the conclusion that some factor in addition to the anoxemia is concerned in the production of anginal pain making it impossible to predict accurately whether or not induced anoxaemia will cause pain. They found that out of a total 56% only showed significant cardiograph changes and their findings agree with those of Sherf & Boyd (55) in laying considerable stress not so much on the actual ST change as on the contour of the ST segment. Many of the cases with angina show a flattening of the main portion of the segment with a quick return to the isoelectric line similar to coronary disease and they maintain that where this is found in spite of the depth of the deviation coronary insufficiency may be presumed to be present. The important feature is that such changes do not appear in the normal but we must bear in mind that certain

slight physiological changes can occur. Wood[58] and his associates found little change in the QRS complex in healthy subjects after exercise except when carried to the point of exhaustion, some right axis deviation then occurred. In the normal he found an inverted T wave tends to become upright while an upright T wave becomes increased in height, but in no control subject did exercise produce a deepening of an inverted T wave or a deviation of the ST segment from the isoelectric line.

In attempting to apply such tests in general practice we are faced with the difficulty that any induction of anginal pain merely for records is quite unjustifiable but a slight degree of exercise short of pain production can in some cases give some results. It is worth remembering that these changes are a measure of the coronary circulation and not of the angina pectoris.

Levy quotes Housner & Sherf[59] who maintain that the RST displacement may outlast the chest pain and can be induced by an amount of exertion insufficient to produce it. Here therefore is an indication of how we can apply this test in private practice without necessarily inducing an anginal attack.

Very recent work on this subject has been an attempt to standardise the amount of exercise required and Master, Friedman & Dack[60] have published the results of their investigations using a fixed number of steps in

accordance with the patient's age, sex etc. This seems a very sound method of approach and appears to me a real attempt to make this test of definite value, but still is fraught with some danger. Their findings are regarded as positive if:-

1. The ST segments are depressed by more than .5 of a m.m.
2. Change from a previously inverted T to a flat or upright T.
3. Occasionally multiple premature beats or wide QRS or deep Q wave.
4. Prolonged PR interval or heart block.

They summarise their findings after investigation of a series of cases in the following manner:-

1. Depression of RST segment.
2. Lowering of inversion of T wave similar to anginal attack or in acute coronary insufficiency such as hemorrhage, shock etc.
3. No significant elevation of ST is observed
4. Changes unlike occlusion since no Q waves appear
5. With few exceptions no reciprocal ST changes in leads 1 and 111
6. Pain during test does not matter
7. Changes last only a few minutes up to 8, sometimes only 1 minute.
8. T.P. interval shortened (diastole shortened due to exercise).
9. May get U wave.
10. Depression of .5 m.m. of ST does not appear much (PR interval taken as isoelectric) but never appears in the normal.

In applying these tests in practice the amount of effort was so graduated as to avoid the production of pain and was based on one group of 20 movements or double this if the case happened to be of a mild nature. The majority of results were found to be indecisive, consisting mostly of depression of the T wave or ST segment in one or more leads; particularly in lead IV. It seems that this is the least decisive change occurring in this condition, but nevertheless T wave or ST depression did not occur in several normal records.

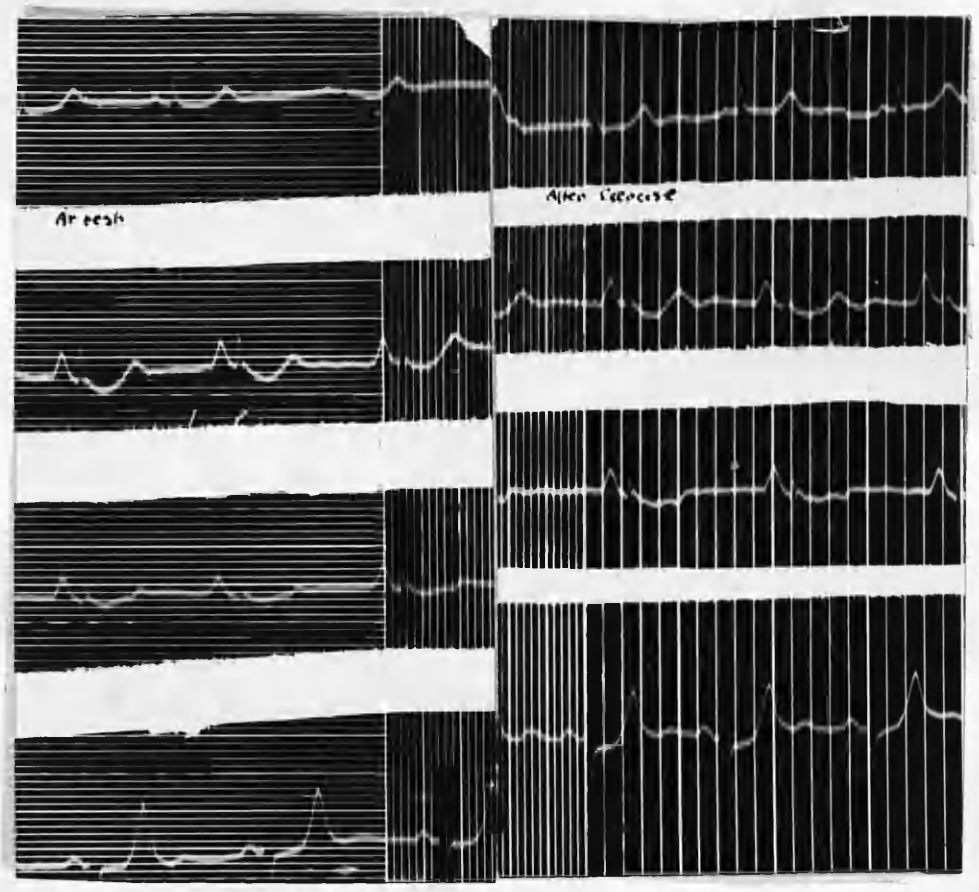
These tests were conducted with the patient on the couch and with the electrodes in 'situ', eliminating therefore any changes due to altered position of patient or connections but not of course allowing for diaphragm changes of position.

The application of the exercise test was however found to be disappointing in actual practice. This may be due partly to the fact that all the cases seen in this series were investigated in general practice and any attempt "to experiment" in such a way as to induce pain or perhaps the danger of an occlusion is to be deprecated in private practice and in fact at any time. For that reason the amount of exercise was never carried (except in a few cases) to a degree sufficient to induce precordial discomfort.

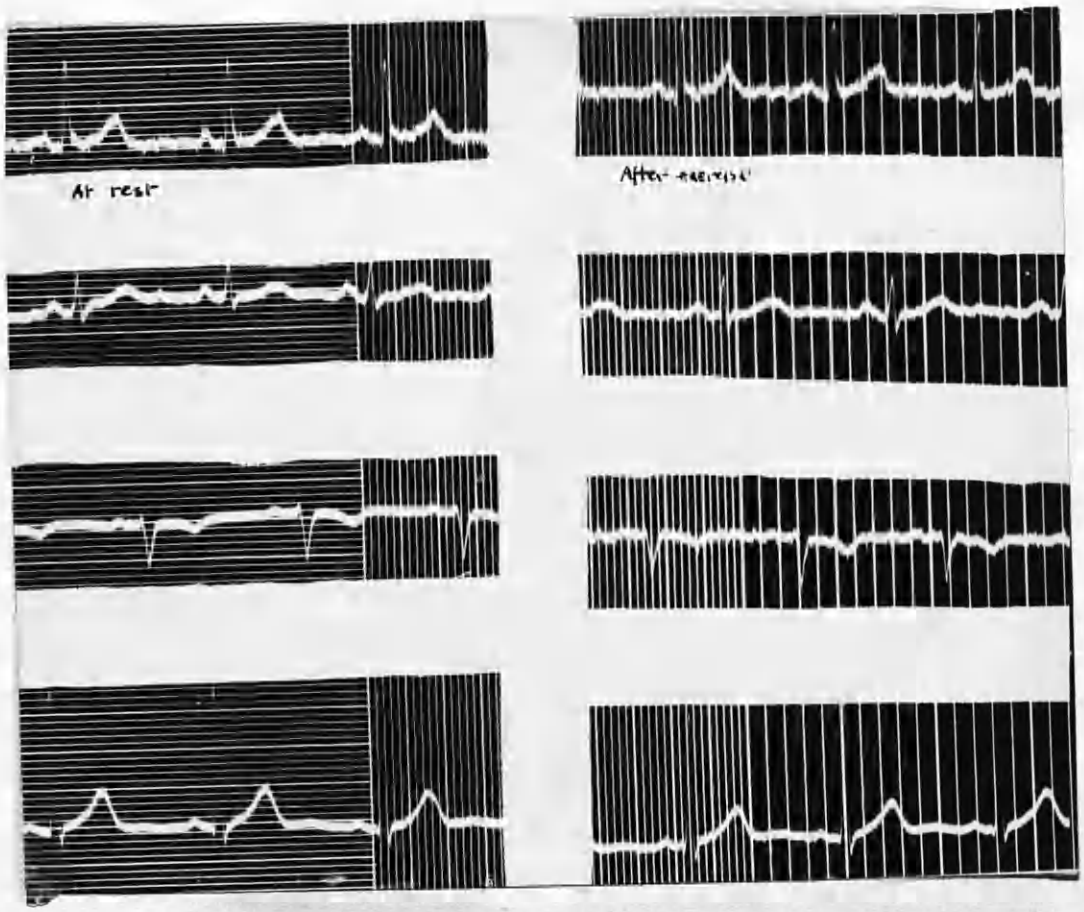
It was found that no constancy of results could be obtained from these tests and many cases with an undoubted history of coronary insufficiency were found to produce entirely negative results and cases which did shew positive changes were already shewing diagnostic features on the electrocardiogram even at rest, but in no case were these changes able to throw much light on a doubtful history.

The following case was an example of advanced anginal syndrome with pain on walking 80 to 100 yards of several years duration, and now shewing considerable diminution in exercise tolerance. Here the ECG at rest shewed pronounced depression of ST2 and ST3 with high pointed T waves in leads 11 and 111. This man has aortic regurgitation with general enlargement of the heart - the P waves no doubt being an indication of auricular enlargement. A low voltage QRS and T in all leads is suggestive of considerable myocardial damage, and a high pointed T4 wave (upright but of coronary type is suggestive of some coronary disease.
On exercise some slight depression of ST2 and ST3 is noted with diminution in T4 and increased prominence of the U wave in the chest lead.

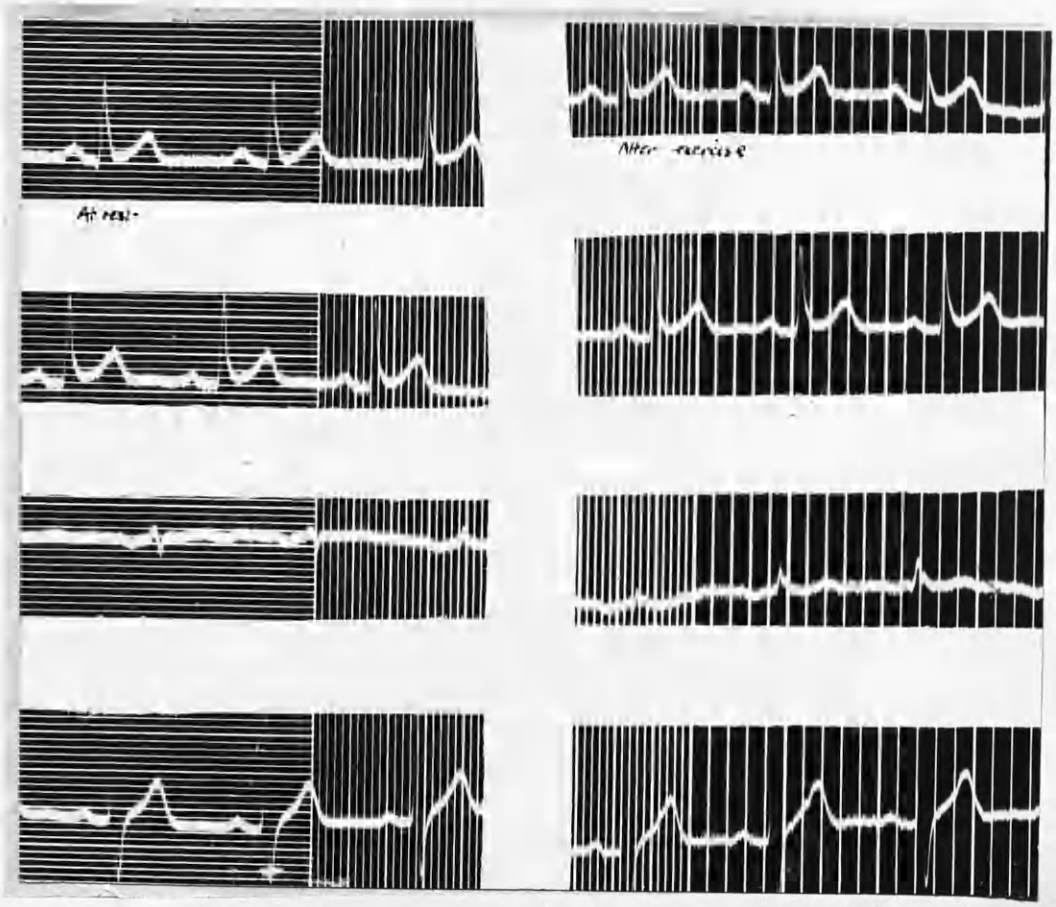
The most significant change here was the depression of ST4 of 1 mm on exercise. This appeared in two seperate tests on the same patient and was never observed in any normal record. In this case some precordial discomfort was present during the test.



The following case was an exercise test applied in the case described on page 146 . Here the clinical history and electrocardiogram gave no support to a diagnosis of coronary insufficiency but the type of T wave inversion in Lead lll although not characteristically coronary was worth investigation since this Pilot was proposing to fly again at high altitudes, sometimes without oxygen. The findings here were negative.



The following records were taken in a case with precordial pain following pneumonia where the clinical findings again, although negative, suggested the possibility of some coronary disease. The pain occurred on exercise but was most pronounced on getting up in the morning and tended to disappear as the day wore on. Although the history is strongly suggestive of some condition such as myofibrositis of the chest wall an exercise test was applied, but no significant change was observed.

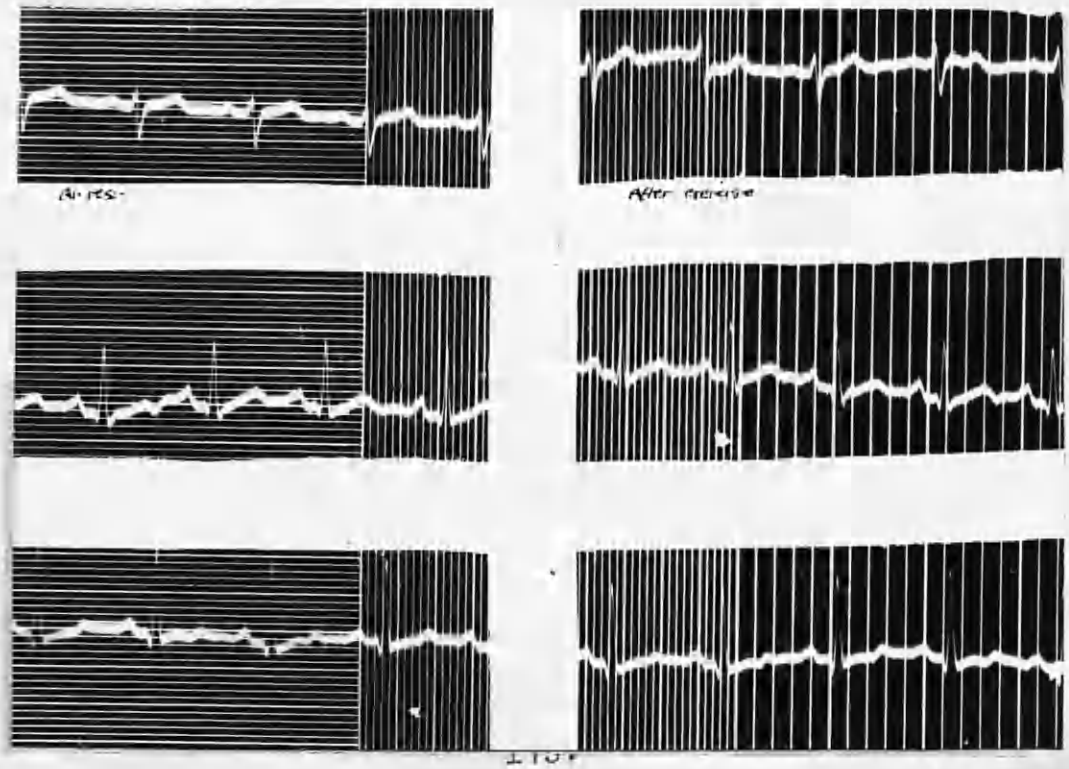


It would appear as the result of these cases and others, that this test is still too vague to be of any practical value from the point of view of everyday use and we are justified in concluding that the degree of coronary insufficiency is not directly related to the degree of pain produced, and no doubt, many cases of coronary insufficiency exist where no discomfort is experienced. This is borne out by the frequency with which an acute coronary occlusion occurs in the absence of any previous history of angina of effort, and where

coronary disease is subsequently found to be present. No doubt this factor is the susceptibility of the individual to pain.

The following was a case of definite clinical history of anginal pain -

Mr. H. Aged 40. Seen complaining of pain on exertion of five years standing but becoming more obvious the last two years since taking up more strenuous life of employment as knife grinder. This pain is left submammary in type but appears to come on with walking and is relieved by rest. An associated pain on outer aspect of left shoulder appears with these attacks.
On examination no abnormality was observed
B.P. 110/80 with some degree of tachycardia
Rate 100
This patient had pronounced kyphosis
The radiograph is shewn on page 184.
ECG shews right axis deviation no doubt in this case due to abnormal position of the heart.
On exercise short of pain production no significant changes were recorded.



As the result of a series of such exercises it seemed that as a practical method of investigation these tests were of little value. No doubt in Hospital where time might be available for repeated tests at varying intervals following the exercise their value might be considerably enhanced but in General Practice the utility of this method of investigation was not proved to be great.

Most changes occurring were of a minor nature and consisted of slight depression of the ST segment in the limb leads or with slight lowering of the T waves but in no instance did an isoelectric T wave or positive T wave become negative. The chest leads shewed the most definite changes and in a few cases definite depression of the ST segment was noted up to 1 millimetre or more with obvious lowering of the T wave. Increased prominence of U waves was noted in several cases, although the full significance does not yet appear to be understood.

It would appear that one factor which might negative the value of such tests would be the position of the diaphragm following exercise and it is maintained by Roesler (71) that the diaphragm does not return to its normal position for a considerable time after exercise.

EFFECT OF ATTITUDE IN CORONARY INSUFFICIENCY.

The whole question of coronary insufficiency is going to be one of vital importance in the future, particularly now that high altitude or stratosphere flying is becoming the recognised method of air travel. The aptitude of certain individuals for flying at these altitudes naturally varies considerably, and depends among other things on the capacity and integrity of the coronary circulation. No doubt the big commercial lines of the future will provide a hermetically sealed cabin with a constant supply of oxygen in the right concentrations and what is equally important at the right temperature. Nevertheless for some time to come, when the public embrace flying as general means of transport such elaborate machines will not be available, and in all probability enormous altitudes requiring sealed cabins may not be resorted to. It will therefore be a matter of the utmost importance to be able to assess the capacity of the individual for high or moderate altitudes and no doubt a great deal of work is being done to-day in our services which must for obvious reasons remain unpublished. We have for these reasons to turn to information available mostly in the United States if we wish to consider the effects of high altitude on

cardiac conditions.

(61)

Experiments conducted by Capt. M.S. White, U.S. Air Arm, Texas, make very interesting reading, and show how early attempts were made by means of rebreather apparatus to simulate the anoxia experienced at high altitudes. The changes occurring in the electrocardiograph on both the normal and abnormal individuals were noted with a view to determining the presence or absence of heart disease. A study of patients with anginal disease receives special attention and it was found that varying degrees of anoxia precipitated attacks of anginal pain, with findings similar to previous workers. It was ascertained for example that definite depression of the ST segment and inversion of the T wave in Lead 1 and IV F should be used as an index of coronary insufficiency alone. These studies were carried out during relatively short period of oxygen deprivation simulating in degree the conditions obtaining while flying at high altitudes. Furthermore, in order to confirm these findings a series of actual tests were performed and electrocardiograph records made in actual flight and at different altitudes and different rates of climb.

One group made the ascent to 15000 feet without oxygen and the other group with oxygen. Records were made on ground level, and at various altitudes in constant temperature, the plane being heated and maintained at

that temperature during the flights. The conclusions were

1. 45 normal subjects remaining at 15000 feet for a two hours period without oxygen showed definite ECG changes mostly decrease in voltage of T wave in all leads and increase in rate.
   This was first noted at 5,000 ft. becoming progressively greater with altitude.

2. A slow rate of ascent slightly lessened the changes

3. After continued flight at 15000 ft. for two hours an apparent compensatory mechanism operates with slowing of the heart rate and increased voltage of T wave

4. U waves developed in slightly more than one half of the cases increasing with increased inoxia.

5. All changes were reversed by the administration of oxygen.

6. Support is given to the theory that myocardial anoxia is a contributory factor in the signs and symptoms of coronary artery disease

As a result of these experiments it was suggested that oxygen should be applied for all flying personnel over 5000 feet but 7500 is suggested as a practicable level for general application of oxygen.

A study by Graybiel et al (62) on the effects of high altitude on cardiac patients is worth consideration. Graybiel summarises his findings in the case of 13 known cardiac cases taken to altitudes up to 14500 feet without oxygen. Few patients complained at all up to this level except 3 who fainted. The pulse rate was

the same as in control subjects but the electrocardiograph showed pronounced decrease in T wave with slight QRS and PR changes. He came to the conclusion that cardiac patients were definitely endangered only if the oxygen concentration fell below 12% that is above 14500 feet.

It is interesting to observe that information reaching us from Germany since the war shows their research along similar lines. The American Aviation Journal quotes the work of Albers D & A Koch[63] in their study of electrocardiograph changes at high altitudes and showing similar results. They found that under 9000 metres ST changes were not pronounced and lowering of the ST interval appeared in about a fifth of the cases. They also found decreased voltage of T wave with increasing altitude especially above 5000 - 6000 metres and mostly in lead lll. They point out that changes in the ST interval or T deflection demand that a person be treated with a certain amount of caution and frequent observation.

Interesting work also has been done by Luft U.[64] demonstrating a process of adaption taking place in the cardiocirculatory system at high altitude. He found that repeated ascents showed increased tolerance to altitude and prolonged stay on high mountains, for

example, caused more pronounced and definite tolerance to altitude. Increase in pulse frequency in short ascents was noticed after 4000 - 5000 metres, but where pilots had lived for some time at great altitudes such as mountaineers do, the increase in rate only occurred over 6000 metres and that some definite gain in altitude tolerance can be achieved by adaption.

X RAY FINDINGS.

The examination of the heart by fluoroscopy and radiography can sometimes give additional information in coronary insufficiency although in this particular condition the X ray has a limited value. Perhaps the most important and frequent changes observed are those associated with hypertension or arteriosclerosis.

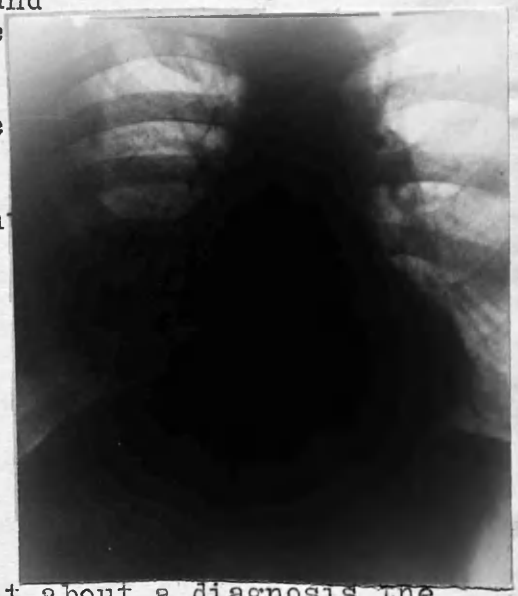
The recognition of these changes can occasionally be of supreme diagnostic importance in cases where a previous high blood pressure has given place to a low one following failure or myocardial infarction. In other known cases the degree of cardiac involvement can often be of great prognostic value.

The so called aortic configuration or "Coeur en Sabot" shewing enlargement of left ventricle with accentuation of the normal concavity of the left ventricular border and wide vascular pedicle can be

frequently recognised. The following case might be regarded as a typical example of hypertensive heart disease in a patient with a history of myocardial infarction two years ago and now shewing angina of effort.

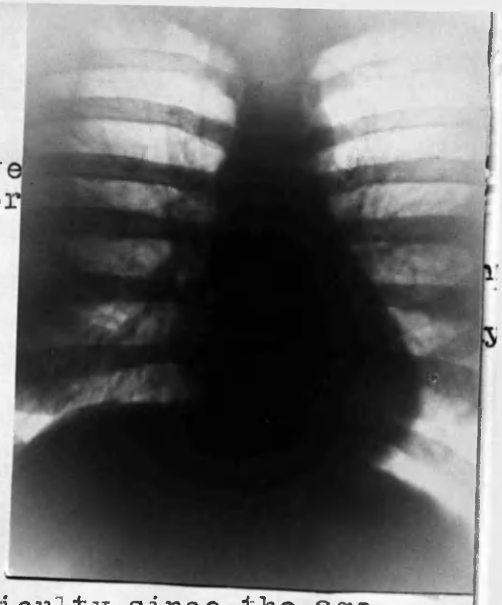
This patient is already described on page 122

Here a prominent left ventricle is seen with obvious convexity of the right border of the ascending aorta, with prominent aortic knuckle and increased density of the aortic shadow. There is considerable elongation of the aorta and obvious unfolding of the aortic loop with prominent descending aorta seen alongside the ascending vessel. This patien is hypertensive B.P. 180/100 with a family history of hypertension. One dominant feature of these records is the apparent curvature of the whole vascular contour due mostly to the convex prominence of the Rt border of the ascending aorta.



In some cases where doubt is felt about a diagnosis the findings on X ray can support an existence of hypertension or arteriosclerotic vascular changes. The following case is that of a woman aged 40 with a history of substernal pain and tightness in the chest occurring on walking quietly.

This patient gives a history of seve
pain four months ago persisting for
several days and which appears
to have marked the commencement
of her pain on exertion. She
gives a family history of her
mother with "anginal" attacks
and high blood pressure."
The B.P. here was 140/100
and the ECG shewed low voltage
QRS and T waves in all leads
with some left axis deviation.



This case presented great difficulty since the age and sex of the patient were naturally against a finding of coronary disease but the X Ray findings shewed a prominent aorta but with a filling in of the normal concavity of the left cardiac border with prominence of the left auricle suggesting general enlargement of the heart.

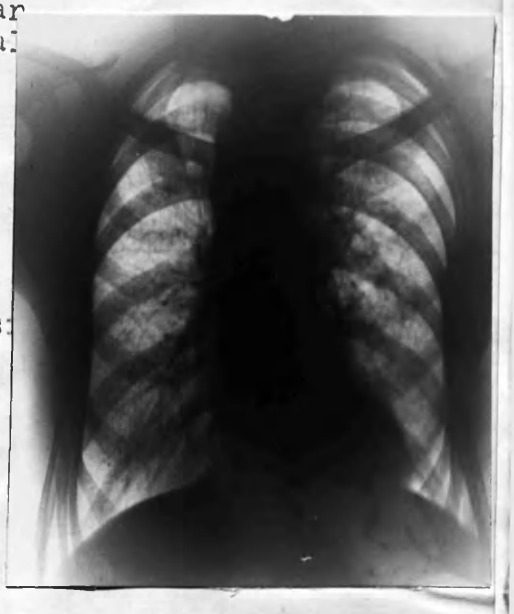
On the other hand the diastolic pressure at the upper limit of normal suggest the possibility of an arteriosclerotic background which is supported by the changes on X ray of the aortic shadow.

The so called hypertensive changes are found in aortic stenosis or regurgitation in which conditions the anginal syndrome is frequently encountered although the diminished prominence of the aorta in aortic stenosis can be of assistance. Calcification of the aorta and even of the coronary vessels can occasionally be observed in support of a diagnosis of coronary disease

but this is a rare finding.

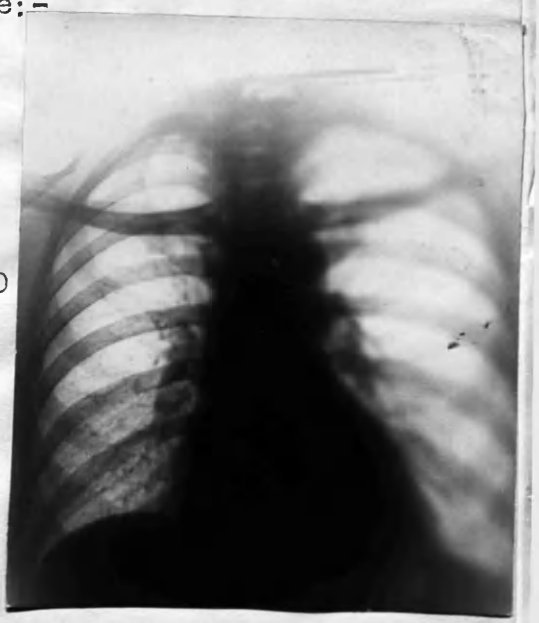
The possibility of deformities of the thoracic cage associated with pressure or displacement of the heart giving the anginal syndrome is worth consideration

The following case is that of a mar aged 45 complaining of substernal pain on effort of six month's duration.
B.P. 150/90
Here a considerable degree of kyphosis exists with funnel shaped chest and a greatly elongated heart shadow.
This patient is a bronchitic subject and shews increased dens of normal lung striation.
The wide vascular pedicle is obvious and it is questionable whether the coronary insufficiency in this case may be due to impaired coronary circulation as the result of cardiac displacement.



The persisting substernal ache of hypertension without definite indication of coronary insufficiency could be illustrated by the following case:-

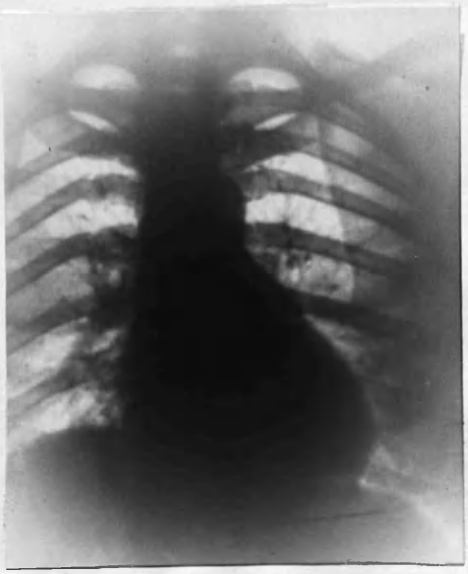
This is a case of a woman aged 49 years with hypertension of some years standing who complains of pain across the chest often for days at a time. This is aggravated by effort but is present even on resting The B.P. was found to be 240/120 with accentuation of second aortic sound. The radiograph shewed a prominent left ventricle and increase of vascular pedicle and prominent aortic knuckle.



Some cases of hypertension do not shew such changes and in fact may have a normal cardiac contour. This may be explained as Roesler suggests by the fact that many such cases are only at the stage of paroxysmal hypertension or perhaps we may be dealing with hypertension in a normally small heart; but generally speaking the above findings are useful as additional evidence.

One type of case can cause confusion here, namely the high diaphragm with apparent enlargement of the left ventricle due to upward displacement of the heart, producing a transverse lie of the whole organ. The following case is such an example:-

This patient aged 45 complains of some pain of supramammary type on exertion and frequent attacks of "palpitation".
B.P. 120/80 Rate 100
Knee jerks were exaggerated with a fine tremor of hands.
The ECG here shewed an isoelectric T3 with inversion of P3 and pronounced left axis deviation, no doubt due in this case entirely to the abnormal position of the heart and not to hypertension, and this was confirmed by X Ray, shewing high diaphragm.



In the recognition of extra-cardiac causes of chest pain the X Ray can be invaluable. In such conditions as pericarditis, thoracic tumours etc. X Ray findings can throw considerable light in certain cases of doubt.

Other evidence of old cardiac injury can sometimes be recognised in the form of cardiac aneurism, aortic aneurism etc. but it must be remembered that enlargement of the aorta can occur in aged individuals without any arteriosclerosis (Roesler). (72)

C O N C L U S I O N S.

A series of cases was taken as the basis of this investigation and the predominant clinical features in each were noted. In all, cases, electrocardiographic records were made and some cases were chosen also for fluoroscopy. An attempt was made to correlate the clinical, electrocardiographic and fluoroscopic findings and to ascertain what outstanding features of each mode of investigation could be regarded as of diagnostic importance. These cases were chosen because of some similarity of the clinical history to the accepted findings of coronary insufficiency and in some cases of doubt electrocardiographic records were made before and after exercise.

The characteristic feature of the anginal case was found to be primarily the location of the pain. In most cases this was found to involve the substernal or upper epigastric region at some time in its course. Radiation into the arms, neck, leg or jaw was by no means a constant feature and the centripetal type of pain was a rare finding. Transient coronary insufficiency seemed to be associated with substernal oppression of

discomfort only and radiation into the arms was found more frequently where a myocardial infarction had complicated the picture. This sense of constriction over the centre of the chest was found to be a very constant feature. The sudden onset of anginal pain on effort without any dramatic incident was found not infrequently to be a premonitory indication of an approaching myocardial infarction.

The association of pain with effort usually involving walking was found to be characteristic of chronic coronary insufficiency and where the pain was induced by arm movements only, one was more inclined to suspect an extra-cardiac cause although this was not invariably the case. The duration of the pain was found to vary within very wide limits and cases giving a history of pain persisting over an hour or more sometimes found to shew no evidence (recent or later) of infarction.

Many cases of chronic coronary insufficiency were found to originate in an incident suggestive of an acute coronary occlusion but equally in some cases no such accident can be found to have occurred although the onset may have been sudden. Cases of myocardial infarction were found to occur most frequently in the older patient while at rest and those cases where this followed excessive exercise were rare and were usually found to be more benign.

Many cases of myocardial infarction were found to shew pulmonary congestion on the left side only, particularly on the pleuro-pericardial margin of the lung with little or no basal congestion and with no obvious pericarditis. This was observed in several cases and the explanation does not seem clear. It was assumed that this may be due to compression of lung tissue along the left cardiac border and is obviously not due to failure which when found produced bilateral basal congestion in some cases. Abnormal location of pain for some weeks prior to the onset of an infarction was observed in a small number of cases. This was found mostly to be pain on one or both sides of the neck.

Examination of the heart in coronary insufficiency was often found to be of little help although certain findings were of assistance. The presence of aortic disease either regurgitation or stenosis was found in a small percentage of cases and occasionally a systolic mumur at the apex was heard. Generally speaking the findings are negative but an accentuation of the second aortic sound can often be accepted as indication of hypertension and was found in some cases where the systolic pressure was low. Cases were found where this sign persisted after a fall in blood pressure following a myocardial infarction. Occssionally a split first sound or gallop rhythm was ascertained but this was a rare finding. The sudden appearance of pronounced rhythm disorder was found in

one or two cases to be associated with myocardial infarction.

Hypertension was a significant finding and where present was usually found to be associated with a poorer prognosis, particularly if significant changes of advanced hypertension and its effect on the heart appeared on the electrocardiogram. Some cases were presumed to be hypertensive where accentuated aortic second sound was heard or the aortic configuration appeared on fluoroscopy. Prolonged anginal pain associated with low systolic and often low diastolic pressure was a frequent finding in acute myocardial infarction, and some known hypertensive cases were found to have low blood pressure readings two and three years after a myocardial infarction. It seemed that a small percentage of cases gave a clinical history of acute infarction with no significant findings on the electrocardiogram. This occurred in cases where the general disturbances were mild and it was presumed that these infarcts must occur in an area which does not shew on the electrocardiogram.

Sedimentation rates were taken in only a few cases since it was felt that this was not a definite diagnostic feature.

Electrocardiographic findings.

Over 50% of cases with the anginal syndrome were found to have electrocardiographic changes of a definite

nature. Included in this group were cases of acute myocardial infarction with recent or late changes. Left axis deviation was a frequent finding but was only accepted as significant if the stage of pronounced left ventricular preponderance with high voltage QRS or the later stages of ST depression in leads 1 and 11 with inversion of T1 and T2 were found. In the latter group of cases protracted coronary insufficiency was assumed to be present and where widening of the QRS beyond .10 of a second was noted the rate of deterioration in the patient's exercise tolerance was found to be rapid. When this widening was associated with notched QRS this was found to be more pronounced, and where left ventricular hypertrophy was associated with aortic disease the outlook was considerably less favourable than where these changes were associated with hypertension alone. A high proportion of cases in the later age groups was found to shew various degrees of intraventricular block or bundle branch block. Here again a wide QRS with significant notching was found to be associated with dyspnoea and a rapid deterioration of the condition. Left bundle branch block seemed to be the predominant finding in this age group, and no case of right bundle branch block was recorded. Since many of these cases had a history of hypertension this appeared to lend support to the theory that bundle branch block can develop from the advanced hypertensive electrocardiogram,

and may not be due in all cases to a lesion of the conduction system. Here again significant notching was usually found to be associated with considerable dysponoea. Generally speaking notching or slurring of the QRS if found in leads 1 and 11 was more frequent in the advanced anginal case.

Low voltage of the QRS was found in several cases following infarction but was usually associated with some other definite electrocardiographic change.

The finding of the coronary T wave was considered of great diagnostic importance and was present in many cases giving the anginal syndrome, but not infrequently a high pointed T wave only present in the chest lead was found in cases where a past infarction could be presumed. Many cases shewed coronary T waves as a permanent and stationary feature often without any obvious history of an acute occlusion. This was particularly so where the voltage of such waves was not high and it was presumed that such T wave changes can occur as a manifestation of a chronic process of healing and decay in the underlying myocardium. This same condition obtained with low T waves particularly in Lead 1 and although repeat records shewed an isoelectric T wave to become positive some cases retained this as a permanent feature. It was therefore assumed that an isoelectric T need not be a manifestation of an acute infarction but

may represent chronic coronary insufficiency. An inversion of T3 was only regarded as being significant if it was definitely of the coronary type, and then preferably in conjunction with some other feature such as inversion of T2. Cases of known myocardial infarction with permanent inversion of T waves of the coronary type were observed over a period up to three years and it appeared that where such changes had remained the patient was more prone to the anginal syndrome than in cases where the electrocardiogram had returned to normal. This did not appear to agree with the accepted findings of some authorities on this subject.

A prominent Q wave was found in several cases of the anginal syndrome and was regarded as a significant finding indicative of old infarction and where a wide Q wave appeared the symptoms seemed to be more rapidly progressive. An absent R wave in the chest lead was found in a few cases where no other indication of infarction was obtained beyond the clinical history.

Disorders of rhythm were found to produce coronary insufficiency and in this group paroxysmal tachycardia and simple tachycardia were frequent. One case of sinus bradycardia with substernal pain on exertion was found. Conversely certain disorders of rhythm were found to occur in coronary insufficiency and were probably the result of

diminished myocardial nutrition. In this group multiple ectopic beats flutter or fibrillation could be included although only paroxysmal fibrillation was found to be associated with pain.

Various degrees of heart block transient or permanent were also found in known cases of chronic coronary insufficiency.

In those cases where no significant changes appeared on the electrocardiogram but where the clinical history was suggestive of coronary disease an attempt was made to ascertain the changes occurring after exercise. This was found to be disappointing since no constant changes were obtained. Some cases shewed definite depression of an upright T wave associated with ST depression of .5 to 1 millimetre in extent. This occurred in some cases with little or no discomfort whereas on the other hand some cases giving pronounced discomfort shewed no significant changes. It was felt as the result of a short series of such experiments that pain is not of necessity an indication of the degree of myocardial ischaemia and that some other factor undoubtedly operates. This no doubt is the sensitivity of the individual to pain. This test was also handicapped by one important feature namely the inadvisability of carrying such tests too far and thereby inducing a myocardial infarction.

This whole factor of the patient's sensitivity to pain appears to negative the value of graded exercise according to height, weight, etc., as applied by some authors in this sphere.

X RAY FINDINGS.

Generally speaking fluoroscopy is not of such value in these cases as electrocardiography but it can nevertheless be very helpful. Perhaps its greatest use in this type of case is in the recognition of the hypertensive heart where no such findings appear clinically. Here a broad wide vascular pedicle with tortuous aorta and prominent aortic knuckle coupled with signs of left ventricular enlargement can often be an indication of past hypertension. Increased density of the aorta with diminished pulsation can be helpful in supporting the diagnosis of arteriosclerosis although it must be remembered that some cases of hypertension can shew a long narrow heart with no obvious enlargement of the left ventricle. In the left oblique position the wide aortic window with overlapping of the vertebral column by the left ventricle can often support the presence of hypertension or aortic disease. Calcification of the aorta or even of the coronary arteries can be very rarely seen. Occasionally the presence of cardiac aneurism following an infarction can lend support to a previous history of infarct.

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