THE DIAGNOSIS and PROCESS of HEALING of MYOCARDIAL INFARCTION - an ELECTROCARDIOGRAPHIC STUDY

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SECTION I - METHOD and MATERIAL

The investigation which is reported in the following pages was undertaken with the purpose of, firstly, comparing the standard limb leads on the one hand with the unipolar limb and praecordial leads on the other, in the diagnosis of myocardial infarction and, secondly, of assessing in what measure the unipolar leads furnish information with respect to the exact localisation of the infarction and to its process of healing.

The work was begun in 1946 in the Cardiology Department of Glasgow Royal Infirmary. The patients were either in-patients or out-patients throughout the period of observation, or they were followed up as out-patients after dismissal from the wards. In most cases it was possible /

/ possible to obtain serial electrocardiograms at widely varying intervals, covering a maximum period of two years. The electrocardiograms were taken with a Cambridge fixed or a Cambridge portable electrocardiograph. The tracings were taken in the customary order; viz. standard limb leads, six or seven unipolar praecordial leads and finally three unipolar limb leads. The positions of the exploring electrode on the chest were those recommended by the Committee of the American Heart Association and Cardiac Society of Great Britain and Ireland in 1938 and 1943. The chest electrode consisted of a small metal disc applied by suction to the chest. Wilson's central terminal was used, without any electrical resistances, intervening between the central terminal and the limb electrodes. The unipolar limb leads were taken after increasing the sensitivity of the instrument so that one millivolt was equivalent to fifteen millimetres. This was the method adopted at the beginning of the investigation, so that, although the Goldberger method of augmentation of the unipolar limb leads became the routine procedure in the Cardiology Department, it was not considered desirable to change the established practice in the present work. Care was taken to ensure that the posture of the patient was unchanged throughout any serial tracings. As a general rule, in-patients were lying flat in bed, unless orthopnoeic, and out-patients were /

/ were invariably in a sitting posture, unless specifically stated to the contrary, digitalis was not exhibited.

The series consists of fifty-nine cases, all but one being males. Age distribution is as follows:

30 - 39 years:	3 cases
40 - 49 years:	19 cases
50 - 59 years:	29 cases
60 - 69 years:	8 cases

The allocation of cases according to site of infarction is as follows:

anteroseptal:	15 cases
anterolateral:	11 cases
posterior (including posterolateral):	28 cases
anteroposterior:	3 cases
lateral:	2 cases

SECTION II

HISTORICAL REVIEW

It would be idle to speculate on what anatomist first described or depicted the coronary arteries. They are clearly outlined in a sketch of the heart drawn by Leonardo da Vinci and dated 1512 and they are figured in the illustrations of "De Corporis Humani Fabrica Libri Septum", published in 1543 by Vesalius, professor of anatomy at Padua. (André Wesal of Belgium). It is believed that the drawings were made by Stephan van Calcar, one of Titian's pupils. The course of the coronary arteries was studied by Vieussens of Montpellier (1641-1716) in his "Traité du Coeur" and pathological states of the coronary arteries, viz. calcification and ossification, were mentioned in the writings of other French and Italian morbid anatomists of the seventeenth century. The association of pathological coronary arteries with dilatation of the heart was recognised by the Italian physician, Lancisi (1654-1720) and also by Senac (1693-1770), who was physician to Louis XV. He was the author of two volumes entitled "Traité de la Structure du Coeur, de son Action et de ses Maladies." While morbid anatomists might also be physicians, they

/ they were not concerned, as far as their writings indicate, with the correlation between clinical symptoms and post-mortem observations. However, in 1761, the subject of pathological anatomy and its relation to clinical medicine received a powerful stimulus from the publication of the five books of "De Sedibus et Causis Morborum" by Morgagni, professor of anatomy at Padua. His records of postmortem examinations are preceded by clinical observations. In the fashion of the times, the writings are in the form of letters to a friend. He describes a case of severe recurrent chest pain in association with pathological coronary arteries and, in another letter, there is a description of coronary arteries, one of which "appeared to have been changed into a bony canal", in a patient who had died of acute intestinal obstruction.

The history of coronary artery disease, in the later decades of the eighteenth century, shifts to the clinical side. In 1772, Heberden, who was in medical practice in London, published his classical description of the illness which he named "angina pectoris" based on notes, written in Latin at the bedside of nearly a hundred patients; his study was entirely clinical. In 1793 there died suddenly, at a hospital conference, the celebrated John Hunter, who, for the long period of twenty years had suffered from angina pectoris. To his former pupil, Jenner, whose fame more happily rests on the discovery of vaccination, fell

/ fell the sombre task of conducting a postmortem examination. He found that the coronary arteries were "in the state of bony tubes", and that two areas on the posterior surface of the heart were "of a white colour -- and covered by an exudation of coagulating lymph." That such would be the condition of the coronary arteries had been the prediction of Jenner and of his lifelong friend Parry, a Bath physician, who was also conversant with angina pectoris. In 1799, Parry published a clinical and pathological study : "An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris; illustrated by Dissections." He was the first to express the view that the ossification of the coronary arteries found in these patients was the essential cause of their angina pectoris and subsequent death.

This theory of the etiology of anginoid pain was shared by the Glasgow anatomist, Allan Burns, who, in 1809, published his "Observations on the Diseases of the Heart." As an experiment he demonstrated that pain develops in the limb muscles, rendered ischaemic by a ligature and he drew an analogy between this pain and angina pectoris. Nevertheless this view was criticised by several eminent physicians. Corrigan of Dublin, in 1837, published a paper"On Aortitis as one of the Causes of Angina Pectoris" and, as late as 1894, Sir Clifford Allbutt was still of the opinion that angina was "the /

/ "the cry of the diseased aorta."

During the nineteenth century there was little advancement in the clinical approach to angina pectoris. In an era where physical signs were the favourite study of physicians it is not surprising that a disease, notable for its paucity of signs, should fail to excite their interest. However the science of cellular pathology was making striking progress in Germany under Virchow and Cohnheim. The myocardium became the object of intensive pathological study. Its various fibrotic lesions, single or multiple, patchy or diffuse, were sorted out; their origin in the ischaemia of chronic coronary disease was acknowledged and their role in subsequent myocardial dilatation and failure was realised. Sudden or acute coronary thrombosis was also recognised. It was regarded as invariably fatal because the coronary arteries were considered to be end-arteries. This conception was supported by Cohnheim's experiments on dogs. He found that ligature of one of the two coronary arteries or a large branch of either was immediately fatal but other workers, repeating the experiments at a later date with improved technique, could maintain the life of their animals for days or weeks. By the end of the century however several postmortem reports were published which described considerable anastomosis between the branches of the right and left coronary arteries and at the same

/ same time pathologists recognised that myocardial infarction or its sequel, parietal aneurysm (as it was called), were originally caused by acute coronary occlusion. These views were clearly set forth in two publications, the first by René Marie in 1896 under the title of "L'Infarctus du Myocarde et ses Conséquences," and the second by Sternberg in 1914, "Das Chronische partielle Herzaneurysma." For the time being the pathological study of the subject had outstripped the clinical approach.

Although the first case of acute coronary thrombosis to be correctly diagnosed during life was described as early as 1878 by Adam Hammer of St. Louis and Vienna it was not until 1912 that in the Journal of the American Medical Association there appeared the celebrated paper of Herrick's entitled "Clinical Features of Sudden Obstruction of the Coronary Arteries", in the course of which he described the clinical symptoms of coronary thrombosis based on six cases, one with postmortem confirmation of diagnosis. Nevertheless, as Herrick himself admits, the paper failed to arouse interest. A similar study in 1910 by Obrastzow and Straschesko in Germany met with the same indifference but a second paper by Herrick in 1918 found physicians prepared to appreciate the importance and apparent frequency of the condition. In his second paper Herrick had added two new cases both /

/ both with postmortem confirmation of diagnosis. Furthermore, he was now using the electrocardiograph and the prospect of enhancing the reliability of the clinical diagnosis was indicated by the resemblance of the tracings of one of the patients to those obtained by Fred Smith in dogs in which a coronary artery had been ligated. Immediately after ligation, he found that the RS-T segment branched off from the R wave above the iso-electric level; within 24 hours the T waves became deeply inverted in all leads and thereafter there was a slow return to the upright form.

Thanks to the inventive genius of Einthoven an electrocardiograph suitable for clinical use was introduced into medicine in 1903. Although Kolliker and Muller noted as early as 1856 that each beat of the frog's heart is accompanied by the production of an electric current, it was not until 1887 that Waller succeeded in demonstrating and recording a similar current in man, by attaching, to the front and back of the chest, electrodes led from a capillary electrometer. In this instrument the variations of potential caused changes in level of a mercury meniscus but incidental physical agents such as friction and viscosity of the mercury militated strongly against the registration of the very small action currents of the heart. Einthoven /

/ Einthoven substituted a string galvanometer of his own design for the capillary electrometer and instead of affixing electrodes to the chest he attached them to the extremities - the right and left arms and the left leg - and thus launched the standard limb leads of to-day. In Britain, Sir Thomas Lewis did much to popularize the electrocardiograph in his Clinical Electrocardiography, published in 1915. Thus the third decade of the century opened with physicians alert to the clinical diagnosis of coronary thrombosis and in the possession of an instrument of precision, the potentialities of which were only beginning to be explored.

During the nineteen-twenties the study of coronary thrombosis was largely electrocardiographic. Amidst the literature, which rapidly accumulated, certain papers are pre-eminent and have become classical contributions to the subject. Pardee (1920) in New York first recorded in man a tracing exhibiting upward displacement of the RS-T segment originally observed by Smith (1918) in dogs immediately after coronary ligation. This deviation of the RS-T segment was recognised as a sign of coronary artery obstruction in man and has since been named Pardee's sign. Pardee (1925) also called attention to the unusual shape of the inverted T waves which followed the disappearance of the RS-T elevation. The descending limb had often a noticeable upward convexity. He named

/ named this pecularity "the coronary T wave"; the same sign was called "the cove-plane T wave" by Rothschild et al (1926). Both labels have passed into common usage. Later, Pardee (1930) made a special study of the Q wave of lead III and provided it was of sufficient size, viz. 25 per cent or more of the largest deflection of QRS, in whichever lead this occurred, it was taken to signify "disease of the left ventricle, so that the right ventricle predominates during the spreading of the contraction." The majority of such large Q3 waves were obtained in patients with angina pectoris, but certain cases of myocardial fibrosis with congestive failure and of rheumatic heart disease especially with pericarditis and occasionally of hypertension gave such records. Pardee recognised that respiratory movements influenced the Q wave of lead III and that a high position of the diaphragm might explain the occasional occurrence of a large Q3 in normal persons. In England, Parkinson and Bedford (1928) published their paper on the sequential electrocardiographic changes following myocardial infarction. They described the sequence of changes in the RS-T segment and T waves, viz. transient deviation of the RS-T segment from the iso-electric plane followed by deep inversion of the T waves in either lead I or lead III and correlated the electrocardiographic sequence with the pathological

/ pathological changes evoked in the myocardium by coronary occlusion - the RS-T deviation indicating spread of necrosis and the T inversion, impairment of function, not confined to the limits of the actual necrosis. They contended that only a large number of serial records, commencing from the time of the attack, could prove that the electrocardiogram had remained unaffected by a clinical attack of myocardial infarction, but they were, at the same time, prepared to admit that there may be areas in the heart which are silent as far as the electrocardiogram is concerned. They concluded with the prediction that the size and distribution of the infarct may decide the lead in which T inversion predominates.

Four years later, in 1932, an anteroposterior chest lead was re-introduced into clinical medicine by Wolferth and Wood (1932a). They themselves pointed out that Waller had used this link-up as long ago as 1887 and that Lewis had applied electrodes directly to the chest in his studies of auricular action from 1909 onwards. The new anteroposterior lead consisted of an anterior electrode placed just to the left of the mid-line at the cardiac level and connected with the right arm wire of the electro-cardiogram and a posterior electrode placed medial to the angle of the scapula and connected with the left arm wire. The normal

/ normal configuration of this lead as originally derived was a diphasic initial ventricular complex beginning with a prominent downward Q wave. The R wave equally large. The RS-T segment had practically no iso-electric period and the T wave was large and inverted. Their clinical work was inspired by their experimental work on dogs (1933) in which they found that an anteroposterior chest lead displayed typical changes in the RS-T segment after ligation of the descending branch of the left coronary artery, while the limb lead electrocardiogram was normal. They predicted that a similar association might occur in the human subject and shortly afterwards such a case presented itself. The patient was a female of 76 years. The diagnosis of myocardial infarction was made with confidence on clinical grounds. The blood pressure shortly after the attack was 180/100; later it fell to 155/90. The standard limb leads on the day of a second, more severe, attack of cardiac pain showed no sign of myocardial infarction but an anteroposterior chest lead showed severe deviation of the RS-T segment. However the standard limb leads do show frank left axial deviation with very shallow diphasic T waves in lead I. The interpretation to-day would be anterior coronary insufficiency requiring further electrocardiographic investigation or early left ventricular hypertrophy /

/ hypertrophy which is now known to be a frequent cause of the absence of diagnostic signs in lead I in anterior infarction. Four days after the first electrocardiogram, lead I showed an elevation in the ST segment measuring 1.5 mm. which the authors admit is "suggestive of coronary occlusion." Thereafter the ST-T segment became flat and later the T wave became inverted. Soon, a second case of myocardial infarction occurred - a male, aged 62 years - in whom typical RS-T deviation was seen both in the standard limb leads and in the anteroposterior chest lead. As evidence of the specificity of RS-T deviation in the anteroposterior lead the authors collected thirty-three controls, viz. twenty normals and thirteen cases of cardiac lesions other than infarction; in none of these controls was there RS-T deviation such as had been found in the two cases of myocardial infarction. This work was published prior to the report on their experimental work on dogs. Soon afterwards (1932b) the same authors published a further paper describing three cases of myocardial infarction in which diagnostic signs occurred in the standard limb leads but not in the chest lead. One of the cases showed a T1 pattern; the other two were probably posterior infarctions which would account for the absence of signs in the anteroposterior lead. The authors conclude that "the purpose of the paper is to /

/ to show that lead IV does not in any way replace the routine electrocardiogram but should be used as an adjunct to it." In the same year, 1933, Katz and Kissin, using the same anteroposterior lead now called lead IV, published the electrocardiograms of twenty-five normals and eleven cases of coronary occlusion of which four showed diagnostic signs in lead IV but not in the standard limb leads. However, lead I of all four cases shows signs which to-day would be considered as suggestive of an anterior coronary lesion, viz. very slight upward bowing of the ST segment and a shallow inverted T wave. Soon, additional chest leads were introduced. Wood et al (1933) added leads V and VI. Lead V had the praecordial electrode applied to the apex or to a point 4 cms. to left of sternum in the 5th interspace and connected, as before, with the right arm wire while the indifferent electrode was the left leg. Lead VI consisted of a posterior chest electrode to which the left arm connection was attached and the indifferent electrode was again the left leg. By using the right arm wire for the praecordial electrode in leads IV and V, these early tracings showed a polarity the reverse of that subsequently accepted. Using particularly chest leads IV and V the authors studied thirty-six cases of myocardial infarction of which nineteen were anterior in situation. They found that the usefulness of these

/ these leads was greatest in the diagnosis of anterior infarction for RS-T displacement was more pronounced and more persistent in these leads than in the standard limb leads. Of the nineteen, six finally came to autopsy; four had infarction of the anterior surface of the left ventricle including the apex and of the anterior half of the septum; the fifth had fibrosis of the left ventricular wall involving its antero-inferior regions and adjacent septum, and the sixth had an antero-lateral aneurysm. However study of the standard limb leads of these patients shows that frequently lead I shows RS and T signs which at the present time would be considered suggestive, if not diagnostic, of anterior infarction. A similar criticism may be made regarding the claims made for lead IV in the diagnosis of coronary occlusion by Liberson and Liberson (1933). In an illustrative case the chest lead showed a conspicuous deviation of the ST-T segment suggestive of an acute myocardial lesion, "where neither the clinical picture nor the standard leads suggest it." However, the validity of this statement may be questioned because the blood pressure fell from 170/90 to 120/66 and the standard limb leads show diphasic T waves in leads I and II with "coving" of their first portions. These authors used the same positions for the electrodes on the chest as had Wolferth and Wood but they connected the anterior

/ anterior electrode with the left arm wire and the posterior one with the right. Thus the main deflections in the chest lead followed the direction of similar deflections in the standard limb leads. They established criteria for the normal chest lead based on twenty subjects essentially similar to those of Wolferth and Wood.

Hoffman and Delong (1933) reported a study of chest leads of one hundred and twenty-five normal cases and a small group of coronary cases. In a subgroup designated as showing normal standard and abnormal chest leads, there are minor variations in the standard leads, e.g. slight inversion of T1, shallow T1 and T2, both of which are suggestive of anterior lesions and both of which would at least warrant further electrocardiographic investigation. In another subgroup where the standard leads were abnormal and the chest lead normal, there are changes of T3 type, clearly indicating a posterior lesion. This relative paucity of signs in chest leads in posterior infarctions is now well recognised. The authors confirmed the observation of Wolferth and Wood that at times lead IV may show abnormal signs before they are clear in the standard leads. They also noted that the chest lead may revert to normal before the standard limb leads and that on the other hand the chest lead may retain a frankly abnormal pattern after the signs in the

/ the standard limb leads have disappeared or at least have become equivocal. Probably the authors' evaluation of the abnormal in limb leads would be considered too exclusive by present day standards.

Goldbloom (1934) investigated twenty-five normal cases and forty ambulant cardiac cases, including thirteen who had had coronary thrombosis. Of the thirteen, four showed an abnormal lead IV "whereas the routine three leads are negative." But scrutiny of the published tracings shows that there are abnormal T waves in all four tracings of such a nature that suspicion of myocardial disease would be aroused.

While most workers favoured the apex-beat as one site for the exploring electrode there was little uniformity in the selection of other sites. However there was general agreement that multiple praecordial leads should be recorded and that the sites for the praecordial electrode should be clearly defined because it was appreciated that slight changes in the position of the electrode caused considerable change in the praecordial pattern. (Hoffman and Delong, 1933; Wood et al., 1933). It was agreed that the right arm or the left leg should be chosen for the indifferent electrode and that positivity of the exploring electrode should be represented by an upright deflection in the electrocardiogram. Groedel (1934) and also Hecht (1936) chose as sites for the

/ the exploring electrode, firstly the apex-beat and, secondly, a point on the praecordium in the fourth interspace just to the left or right of the sternum, the indifferent electrode being placed on the right arm. Master (1934) investigated one hundred and four normal adults, placing the exploring electrode near the lower end of sternum about the level of the apex and slightly to the left of the mid-line. The posterior electrode was placed at the same level on the vertebral column. Later he selected the left leg as the site of the indifferent electrode. Bohning and Katz (1938) used the same arrangement of electrodes, viz. the praecordial electrode placed just to the left of the mid-line in the fourth interspace and the indifferent electrode on the left leg. They studied two hundred cases of coronary disease over a period of three years. Twenty-five of the series were ultimately examined postmortem. The conclusion reached by these workers was that lead IV was of definite value in determining the presence, site and, to some extent, the age of myocardial infarctions, especially those involving the anterior wall. Roth (1935) used three praecordial leads, viz. the right pectoral (sternal end of fourth right interspace) the left pectoral (midway between the right pectoral and the apex); and the apex itself; each was paired with the right arm and the left leg. He favoured the left pectoral lead to which

/ which he invariably attached the left arm wire and thus obtained a tracing of which the principal deflections were in the same direction as those of the standard limb leads. He pointed out that in this lead the initial upward deflection of the ventricular complex was absent in anterior myocardial infarction and that it remained so, indefinitely, as "a residual stigma". A similar observation had been made by Wood et al (1933), who reported disappearance of the initial small component of the QRS complex in leads IV and V, as well as deviation of the RS-T segment, in acute anterior infarction. In their work, the older electrical link-up was customary and the small component was therefore downward. In addition they noted the permanence of this sign. Wilson et al (1932a) had also described the absence of the initial positive wave in anterior infarction in chest leads. Wood and Seltzer (1939) used the same chest leads as Roth did. They studied thirty-three cases of myocardial infarction and found serial standard limb leads diagnostic in all but one instance but in four others the changes occurred earlier in the chest leads so that the diagnosis was made sooner. They derived no help from the chest leads in posterior infarction for, here the limb leads required no confirmation and "the chest leads had little to give."

In 1938 the American Heart Association and the

/ the Cardiac Society of Great Britain and Ireland published their recommendations for the standardisation of praecordial leads. They selected a series of leads from the following points - the sternal end of the fourth right interspace, the sternal end of the fourth left interspace, a point midway between the latter and that next to be described, the mid-clavicular line in the fifth left interspace, the anterior axillary line at the same horizontal level, the mid-axillary line at the same level. These are numbered C (chest) 1 to C6 with a further initial R for right arm and F for left leg according to the site selected for the indifferent electrode. If it consists of the central terminal of Wilson (to be described) the letter V is used. Later, (1943), C7 in the posterior axillary line and C8 in the line of the angle of the scapula were added by the American Heart Association since they were of particular value in the study of posterolateral infarction.

Following the official publication of 1938 several other papers were published establishing the normal variations of all six praecordial leads and also the abnormal patterns in various cardiac lesions. Edwards and Vander Veer (1938) studied sixty-seven subjects of whom ten were normal. Of the six praecordial points, paired with the right arm, left arm and left leg, they preferred lead CR4 because of the greater amplitude of

/ of the deflections in this lead. Deeds and Barnes (1940), on the data obtained from a hundred normals, fifty of either sex, also found the right arm the most satisfactory site for the indifferent electrode; they were less favourably impressed by the left arm and least of all by the left leg as the position for the indifferent electrode. Shanno (1940), studied a hundred normal subjects (nurses from 18 to 22 years) using the left leg as the site for the indifferent electrode.

With increasing experience most workers began to favour the right arm rather than the left leg as the site of the indifferent electrode. When the former was used, the T waves showed less physiological variation. They were invariably upright in adults and only rarely inverted in children. Wolferth and Wood (1940), wrote an ingenious paper predicting the effect of the potentials of right arm and left leg on those of any given praecordial lead by a study of the standard limb leads. They selected the T waves in the first instance since they are simultaneous in all three standard limb leads. For example, if T1 is +4, T2 is +2 and T3 is -2 then T of RA is 2, T of LA is 6 and T of LL is 4. Hence if the right arm is used as the indifferent electrode, the T waves of any praecordial lead will be two units smaller than if the indifferent electrode had had no potential, whereas if the left leg is used as the

/ the indifferent electrode, the T waves of the same praecordial lead will be four units smaller than if the indifferent electrode had had zero potential. In other words, for this T relationship in the standard limb leads, the lesser degree of distorsion of the praecordial T waves is obtained by pairing with the right arm rather than the left leg.

While physicians were preoccupied with the clinical application of praecordial leads and were appraising their diagnostic usefulness at times with unwarranted enthusiasm, the theoretical and experimental approaches to electrocardiography made rapid advancement during the fourth decade of the present century under Wilson and his associates in Michigan. In common with other investigators they used a bipolar praecordial lead, the indifferent electrode being placed on an extremity, usually the left leg, but they made the further advance of eliminating, by calculation, the effect of the potentials of the remote electrode from those recorded by the praecordial electrode; in other words the actual potential of the praecordial electrode was calculable, (Wilson et al, 1931b). These principles were first applied by Wilson et al (1932b) to their classical studies of human bundle-branch block.

Shortly afterwards (1934a) they performed a series of experiments on the mode of excitation of the dog's /

/ dog's heart. An exploring electrode was applied directly to the exposed epicardial surface, the potential variations of which, they pointed out, were twenty to thirty times as great as those of an indifferent electrode placed on an extremity or on some other part of the body distant from the heart. The necessity to free the former from the influence of the latter no longer arose. Thus the potential variations of the exploring electrode, as recorded, were considered to be those actually occurring at that point.

With such leads the electrocardiographic record, obtained from the exposed ventricles of a normal canine heart, shows firstly a positive deflection due to the spread of the excitatory process from endocardium to epicardium at the point of contact of the electrode, the potential of which becomes increasingly positive as the excitation wave approaches it. When it arrives at the epicardium the potential of the electrode suddenly falls to a zero or negative value. This abrupt movement is called "the intrinsic deflection." Sometimes the original positive deflection is preceded by a small. negative deflection which represents electrical forces generated before the subendocardial muscle beneath the electrode has been activated. As a rule the RS-T junction and the RS-T segment are close to the isoelectric level; the T wave varies in sign and in /

/ in size from region to region of the exposed epicardial surface.

Using the same direct leads Johnston et al (1935) and Wilson et al (1934b and 35) studied the electrical potentials over experimental infarcts in dogs. They found that immediately after production of the infarct by arterial ligation there is displacement of the RS-T junction and segment in a positive direction and diminution or disappearance of the intrinsic deflection. Further, a large negative Q wave develops and the final ventricular complex may consist entirely of a large negative monophasic deflection. Absence of the normal initial positive deflection or R wave is due to failure of electrical forces normally contributed by the involved muscle. As a convenient label, this form of curve is called the central type since it is obtained over the centre of the infarct. In leads from the margins of the infarct, the initial Q wave is less conspicuous; the pre-intrinsic R wave and the intrinsic RS deflection are preserved although diminished in size. Such curves are conveniently named "marginal."

The displacement of the RS-T segment above mentioned regresses after several hours in experimental canine infarcts. It is followed by the development of very large inverted T waves which, in experimental infarcts, last not more than a day.

/ As early as 1930 Wilson had suggested that a praecordial electrode would be of value in the study of human myocardial infarction. Having as a foundation the curves obtained in experimental infarction in dogs, as outlined above, he compared with them the patterns obtained by praecordial electrodes in human myocardial infarction. The necessary use of praecordial or, as he called them, semidirect leads in man compared to the epicardial or direct leads in dogs involved some modification of the tracings. The potentials variations of a praecordial electrode are much smaller than those of an epicardial electrode. The potential variations therefore of the indifferent electrode, wherever it may be placed, are relatively much larger and cannot be disregarded. While he might have reverted to a mathematical elimination of the effect of the indifferent electrode from the finished tracing, he evolved at this time (1934), an indifferent electrode of practically zero potential, now well known as the central terminal of Wilson, so that the recorded and actual potentials of the praecordial electrode are practically identical and furthermore that the curves obtained bear a striking resemblance to those obtained experimentally with direct epicardial leads. The first description of the central terminal appeared in 1934; it is based on the following principles. If a single terminal is connected through /

/ through equal resistances to any three electrodes, then the potential of the terminal is equal to the mean potential of the three electrodes. Furthermore, if the three electrodes chosen are at the apices of the equilateral triangle of Einthoven (right arm, left arm and left leg) and if the theory of the equilateral triangle is valid, then the potential of the central terminal is zero and hence if an exploring electrode is paired with such a central terminal, the record obtained is that of the potential fluctuations of the exploring electrode alone (Wilson et al, 1931a, 1934c).

In their earlier work, Wilson and his associates used resistances of 25,000 ohms, one such resistance being introduced between the central terminal and each of the three electrodes on right arm, left arm and left leg respectively. It was at first maintained that the resistances had to be large in comparison with the largest body resistance offered by skin and internal structures between each pair of electrodes. However these large resistances made the apparatus too sensitive to stray electric currents so that 5,000 ohms were subsequently employed. This central terminal remains of zero potential whether the exploring electrode is placed on the praecordium or on an extremity. While the curve obtained with such a unipolar lead placed on the praecordium is very similar to that recorded by a

/ a praecordial lead paired with an indifferent electrode placed upon an extremity this does not obtain when the exploring electrode is placed further from the heart. With the use of the central terminal however, it is possible to record the precise electrical potentials of an extremity or of any point distant from the heart. Wilson's procedure was to take a unipolar lead from each extremity (VR, VL and VF, right arm, left arm and left leg respectively); from five points across the praecordium from right to left (V1: right edge of sternum at level of fourth or fifth costal cartilage; V2: left sternal margin at same level as V1; V3: midway between V2 and V4; V4: midclavicular line or apex-beat; V5: anterior axillary line); sometimes also V6 in midaxillary line, and VE at the ensiform cartilage.

Using this technique Wilson et al made further studies on myocardial infarction in man (1931c, and 1932a). He found that when the anterior wall of the human heart is infarcted, the tracings obtained by praecordial leads are closely similar to those seen in direct leads in experimental infarcts in dogs, as described above. In the early stages there is positive displacement of the RS-T junction and segment, as originally described by Wolferth and Wood (1932a) for their apical lead IV. The ventricular complex may have the form of a single large negative deflection or Q wave especially over the centre

/ centre of the infarct, although occasionally a small R or an RS deflection persists especially towards the margins of the infarct. Sometimes the R wave is represented by a notch near the base on one or other of the limbs of the Q wave. As the RS-T displacement subsides, large negative T waves develop. These were also described by Wolferth and Wood at a previous date. Thus, as Wilson et al pointed out, there is no essential difference between the changes in the ventricular complex in experimental infarction in dogs and those regarded as diagnostic of myocardial infarction in man. The chief dissimilarity is in the time of occurrence and the duration of the changes. In general, the changes take longer to evolve and to retrogress in man. The RS-T displacement may persist for a week or longer and the subsequent T changes may take months to retrogress. However the modifications of the QRS deflection are frequently permanent in man as they are in dogs. They also noted that the changes in the QRS complex and those involving the T wave are not necessarily most conspicuous in the same lead, frequently the latter are best seen in leads further to the left than the former. Wilson recognised the difficulty of interpreting correctly a large QS deflection in leads V1 and V2 and cited a case (1936) which displayed those signs but which at postmortem examination showed only hypertrophy of the /

/ the left ventricle secondary to aortic stenosis.

In another paper from the same school by Kossman and de la Chapelle (1938) absence of the R wave was noted in leads V1, V2 and V3, although, at post-mortem there was no infarction of the free walls of the right or of the left ventricle. By probing and skewering experiments they proved that the right ventricle was actually subjacent to the V2 and V3 positions in many instances. However they found involvement, by infarction, of the left side of the septum in the particular cases studied, and therefore came to the conclusion that the normal R wave of the anterior praecordial leads depended on the preservation of the normal electrical activity of the left side of the septum. In view of the fact that the R wave might be missing in leads from the right praecordium as a normal variant and in the left ventricular hypertrophy, the diagnosis of septal infarction could not be made unless signs of infarction were present in other praecordial leads, e.g. a QR pattern or absence of R waves or by abnormal T patterns not attributable to left ventricular hypertrophy. However these workers laid little emphasis on cardiac position as a determinant of QRS pattern. The explanation of the facts in modern terms is that hearts showing left ventricular hypertrophy tend to be horizontal in electrical axis and that they, as well as normal horizontal hearts tend to have no R wave in /

/ in leads V1 and V2 because the potentials of the ventricular cavities which are negative are referred to the V1 and V2 positions.

Kossman and de la Chapelle were also interested in the preservation of the R wave, although diminished in lead V3 and in those further to the left in the presence of lesions of the anterior cardiac wall. While they recognised that temporary functional reduction of electrical forces could account for such a diminished R wave, they also suggested that it might be due to fibrous replacement of a sufficient number of muscle fibres or a subendocardial infarction of limited degree.

Wilson (1936) also recognised that the value of praecordial electrocardiography is not limited to diagnosis alone. Some assessment of the size of an anterior infarction can be reached from the number of leads in which diagnostic signs appear; if they occur in all five or six praecordial leads, a large infarct can be suspected, or if only in one or two, only a small part of the anterior wall has been infarcted. Sometimes leads V2 and V3 show characteristic signs while lead V4 is negative; in such cases the standard limb leads are also likely to be negative or difficult to interpret and hence, as Wilson pointed out, "it would seem unwise to rely upon a single praecordial lead from the region of the apex beat," which was, in fact, the site of lead IV

/ still be recognised in spite of right bundle branch block. Similarly the RS-T pattern depends upon the relative magnitude of the electrical forces produced by the infarction in comparison with those due to the bundle branch block.

While the nomenclature of praecordial electrocardiography was officially clarified by the publications of the committee of the American Heart Association and the Cardiac Society of Great Britain and Ireland (1938) and subsequently by a supplementary report from the American Heart Association (1943) there was little agreement regarding the merits of the various leads, in particular the zero potential claimed for Wilson's central terminal was challenged on theoretical and experimental grounds by Wolferth et al (1941), and Wolferth and Livezey (1944). Experiments had been performed by Eckey and Frohlich (1938), by Burger (1939) and by Wilson (1946) to detect any trace of potential in the central terminal and to prove that it was so minute that it could be disregarded. The experiments consisted of immersion of a subject in distilled or tap water in a tub or in a fresh water lake. In the first instance the tub was metal-lined and a suitable electrical link-up made between the lining and the central terminal; in the second instance, a large metal electrode was placed in the lake eleven feet from the

/ lead IV, the single praecordial lead originally introduced by Wolferth and Wood (1932a).

On the other hand Wilson (1936) recognised that the praecordial leads had relatively little information to offer in the diagnosis of posterior infarction except during the early stages when they may show depression of the ST segment but the potential variations of a left thigh lead may show positive signs of infarction in such cases because posterior infarcts generally involve the diaphragmatic surface of the heart. The difficulty of diagnosing lateral infarction from the usual sites of praecordial leads was soon evident because positive signs were more prominent in standard limb leads I and II than in the axillary leads V5 and V6. It must be conceded however that the standard of normality by which leads V5 and V6 had been judged was unduly wide by modern criteria, for in the case figured both show negative T waves. Combinations of signs due to two infarctions of different age or due to a continuous anteroposterior infarction were also described as were also the effects of bundle branch block on the signs of infarction. Right and left bundle branch block are both common in the presence of myocardial infarction. Wilson described how left bundle branch block conceals any signs of infarction of the free wall of the left ventricle in the QRS complex of praecordial leads, whereas they can still /

/ the body and the potential variations of the central terminal with respect to this electrode were measured. With the tub, the potential variations of the central terminal were found to be 0.2 to 0.3 millivolts (Eckey and Frohlich), 0.26 millivolt (Burger) and 0.15 millivolt (Wilson). Burger himself expressed doubts as to whether the minute potential variations of the central terminal could be ascertained by such immersion experiments. Wolferth and Livezey (1944) were also sceptical of the method and claimed that an electrode placed on the right scapula was more uniformly indifferent than the central terminal. In Wilson's review of the subject (1946) he states that all the available data which have a bearing on the central terminal are consistent and that all immersion experiments give essentially the same results, which are important considerations in estimating their significance. He himself remained of the opinion that the potential variations of his central terminal did not exceed 0.3 millivolts.

Goldberger (1942) introduced some modifications of the Wilson technique. He dispensed with the 5,000 ohm resistances because of the fairly high skin-electrode resistances normally encountered. This simplified central terminal is used as the indifferent electrode as before, the exploring electrode being placed on the praecordium or on a limb, just above the elbow or the/

/ the knee, as required. The tracings obtained, namely, praecordial and "ordinary" limb leads are identical with both techniques. Goldberger also discovered that he could augment the limb lead tracings by one half if he detached the connection between the central terminal and the limb, the potential of which was being recorded; at the same time he utilised the limb electrode already in situ as the exploring electrode by attaching it to the left arm cable of the electrocardiogram. These "augmented" unipolar extremity leads were designated aVR, aVL and aVF. Since the sensitivity of the galvanometer is not altered he advised that these records be read in millimetres, since their actual amplitude in millivolts was less by one third compared with that of ordinary unipolar leads.

The two main advantages of the augmented method were, firstly, that there was no further need for an additional exploring electrode on the limb and, secondly, the frequently small unipolar extremity potentials were rendered larger without distortion of their forms and without increasing the sensitivity of the galvanometer. However, in 1949, Bryant, Johnston and Wilson of the Michigan school again advocated the use of the 5,000 ohm resistances on both practical and theoretical grounds; they considered that the potential of a central terminal connected to the limb electrodes /

/ electrodes through resistances of 5,000 ohms and the potential of a central terminal connected directly to these electrodes might be expected to differ significantly in about one case out of ten.

In 1949 The British Cardiac Society recommended the general adoption of the unipolar V leads. Such a measure would have had the great advantage of uniformity of technique in different clinics but there were still grounds for doubt as to the best indifferent electrode and at the turn of the century further papers appeared comparing the bipolar chest leads CR, CL and CF with the V leads and also the Wilson with the Goldberger methods of taking V leads.

Dolgin et al (1949) studied forty-four normal adults in whom CR, CL, CF and V leads were taken; the unipolar extremity leads were taken by the Goldberger method but with the 5,000 ohm resistances retained. They found that the size of the deflections was greatest in CR and smallest in CF leads. When the standard limb leads indicated right axial deviation, CF leads showed very small deflections over the left praecordium and, conversely, when there was left axial deviation CL leads showed similar but much less pronounced diminution in deflections over the left praecordium. CR and V leads were less influenced by axial shift. They also subscribed to the old view that the employment of the /

/ the central terminal eliminated the effect of the extremity potentials on the praecordial tracings. They studied several abnormal subjects whose electrocardiograms showed abnormalities of a kind which might not be consistently recorded by all techniques. Of particular interest is a group with healing or healed posterior infarction, diagnosed by inversion of the T waves in leads II, III and VF. This finding is associated with inversion of the T waves in leads V5 and V6 justifying the diagnosis of posterolateral rather than posterior infarction but the CF leads showed upright T waves in all six praecordial positions obviating the diagnosis of involvement of the lateral wall. The CR and CL leads were similar to the V leads.

A similar analysis of C and V leads was published in 1950 by Leatham who succinctly points out that "leaving theoretical considerations aside, the best chest lead is the one which varies least in health and shows the earliest changes in disease." As a result of his examination of a hundred healthy adults, he found that normal variations in CF leads are very great compared with those in CR and V leads due to the great changes in potential at the electrode on the left foot with changes in the position of the heart. He found that for practical purposes the right arm is equally as good as the central terminal; on the one hand the right /

/ right arm has approximately a constant potential and hence the same error is always introduced and on the other, the potential of the central terminal approaches zero, but is inevitably influenced by the variations in potential of the left foot. The normal T inversion sometimes shown by CF leads over the right praecordium and occasionally seen in V1 and rarely in V7 leads is a further disadvantage of CF and V leads respectively. A similar study was undertaken by Cameron (1949) who investigated sixty cases (thirty normal and thirty abnormal) in whom standard limb leads, unipolar limb leads and multiple praecordial leads of the V, CR, CL and CF types were recorded. He found that the V leads represent the mean of the CR, CL and CF leads, i.e. that the distorting effect of the remote electrode is thereby reduced to a minimum.

Side by side with the experimental, clinical and theoretical investigations pursued by various workers and summarised above, efforts were made to establish and interpret the normal patterns of unipolar V leads in particular. Kossman and Johnston (1935) of the Michigan School using the Wilson central terminal examined thirty young male adults and found that the R wave representing a positive variation in the potential of the exploring electrode is invariably present, marking the beginning of a large and rapid /

/ rapid excursion of the string which corresponds to the intrinsic deflection of direct, experimental leads. When timed by a simultaneous standard limb lead I the apex of R is 0.02 sec. earlier in leads over the right side of the praecordium than in those over the left side.

Several years later, in 1944, Wilson et al summarised their views on the interpretation of the praecordial electrocardiogram. They believed that the passage of the cardiac impulse causes positive potentials ahead of it and negative potentials behind it. Both sides of the septum are activated from their endocardial surfaces inwards, the left being ahead of the right. Hence for a brief moment the potential of the right ventricular cavity is positive, and any lead facing into the right ventricular cavity and thus in apposition to the right side of the septum registers a small positive or R wave. Conversely, at the same moment, the left ventricular cavity is negative and any lead facing into the left ventricular cavity and thus also the left side of the septum registers a fleeting negative potential or Q wave. Thereafter the wave of excitation passes from the endocardial surface of the free walls of the ventricles outwards, causing a sudden fluctuation in potential registered by the electrode as a positive or R wave. Wilson et al do not, in this /

/ this article, discuss further this dual explanation of the R wave of leads facing the right side of the interventricular septum and the free wall of the right ventricle. They describe the intrinsic deflection as beginning at the moment when the excitatory process reaches the epicardium and the whole thickness of the wall is activated. At that instant, potential differences in that part of the wall disappear and the electrode suddenly registers the negativity of the cavity below. This sudden drop in potential completes "the intrinsic deflection." As stated above, the peak of R is earlier, by 0.02 sec. in leads from the right side of the praecordium than in leads from the left side owing to the greater thickness of the left ventricle. Leads from the right side of the praecordium have therefore a small positive R wave followed by a large negative S wave; leads from the left side of the praecordium have a large positive R which is often preceded by a small Q wave and followed by an S wave. Between the right and left sides is a transition zone where R and S are of intermediate form. Thus if the endocardial muscle of some part of the ventricular wall passes into activity earlier than the muscle below the electrode, this initial negativity of the cavity is transmitted to the electrode and a Q wave results. If the excitatory process is still spreading through some /

/ some part of the ventricular wall after the excitation of the muscle under the electrode, the negativity of the cavity outlasts the intrinsic deflection and an S is inscribed. In other words the peak of R separates deflections due to muscle activated before, from those due to muscle activated after, the cardiac impulse reaches the ventricular epicardium under the electrode.

By taking unipolar leads from the right arm (VR) left arm (VL) and left leg (VF) Wilson established a relationship between the potential variations of the right and left sides of the praecordium and those of the limbs. When the heart is normal praecordial electrocardiograms are of constant form irrespective of the axis deviation of the standard limb leads but the unipolar limb leads vary greatly with the position of the heart. When the standard limb leads show right axial deviation, the potential variations of the left arm are like those of the right side of the praecordium whereas the potential variations of the left leg are like those of the left side of the praecordium. The reverse occurs in left axis deviation. This relationship is based on the fact that the potential variations of the left arm resemble those of that part of the heart's surface nearest to the left shoulder whereas the potential variations of the left leg are /

/ are like those of the diaphragmatic surface. A similar relationship holds in the case of the right arm but it is opposed to the valvular orifices at the base of the heart through which the negativity of the ventricles is transmitted, irrespective for the most part of the axis deviation. Thus by comparing VL and VF with V1 to V6 various electrocardiographic positions of the heart can be determined (Wilson differentiates six) even when the axis deviation of the standard limb leads is masked by other electrocardiographic abnormalities. The six positions are :-

Vertical Position

a) The ventricular complexes of VL resemble those of leads V1 and V2.

b) The ventricular complexes of VF resemble those of leads V5 and V6.

Semivertical Position

a) The ventricular complexes of VF resemble those of leads V5 and V6.

b) The QRS deflections of VL are small.

Intermediate Position

The ventricular complexes of VL and VF are similar in form and size and like those of leads V5 and V6.

Semihorizontal Position

a) The ventricular complexes of VL resemble those of leads V5 and V6

b) The QRS deflections of VF are small. /

Horizontal Position

a) The ventricular complexes of VL resemble those of leads V5 and V6.

b) The ventricular complexes of VF resemble those of leads V1 and V2

Indeterminate Position

No obvious relationship between limb and praecordial leads.

It must be clearly understood that Wilson's six cardiac positions are concerned with rotation round an anteroposterior axis. They do not take cognisance of rotation around the other two axes, viz. the long axis of the heart itself and a transverse or horizontal axis through the heart, the effects of which may be conspicuous in the electrocardiogram, as will be discussed later in this paper. Furthermore, Wilson made it clear that he used the terms, vertical, horizontal, etc. solely in respect of the electrical position. He did not imply that they could be applied indiscriminately to the anatomical position of the same heart. While the anteroposterior axis is the only one, anatomical rotation about which can be roughly checked radiologically, and while hearts which are frankly vertical on x-ray examination are, as a rule, vertical electrically, and similarly for the horizontal position, yet there are large numbers of hearts which are intermediate or /

/ or average in position on x-ray examination but which, nevertheless, are semivertical or vertical, semihorizontal or horizontal electrocardiographically. These facts are borne out in the following pages.

Goldberger's approach (1949) to normal unipolar electrocardiographic patterns is somewhat different from that of Wilson. He classified them into five groups on theoretical grounds :-

1) Leads which face the epicardial surface of the left ventricle. They show a qR pattern and T is usually upright. They may occasionally have an R or a qRS pattern.

2) Leads which face the epicardial surface of the right ventricle. They show an rS or RS pattern and T is usually upright but may sometimes be inverted. An rSr' pattern is occasionally found.

3) Leads which face the cavity of the right ventricle. They show an rS pattern and T is inverted. Sometimes a QS pattern may appear.

4) Leads which face the cavity of the left ventricle. These have a QS pattern and T is inverted.

5) Leads which face the back of the heart. These have a QR pattern and T is inverted. Sometimes a Qr or a qR pattern may appear.

Goldberger based the above classification on a study of multiple leads over the upper half of the trunk/

/ trunk as well as on the limbs. He divided the trunk by three lines through i) the second interspaces, ii) the fifth interspaces, iii) the eleventh interspaces. At the points where these are transected by three vertical lines, viz. the mid-scapular, the mid-axillary and the mid-clavicular, leads are taken. Thus leads V4 and V6 are included but the other four praecordial leads, V1, V2, V3 and V5 are also recorded, as well as the unipolar limb leads, a head lead and leads from both supraclavicular fossae.

Leads from the right arm, right shoulder girdle anteriorly and posteriorly, head, left shoulder girdle posteriorly and right anterior part of chest, including lead V1 can be said to face either ventricular cavity and therefore their main deflection is negative. Leads overlying or facing the epicardial surface of the left ventricle, i.e. from the left side of the praecordium, left upper abdomen and left lower back have a positive main deflection. Leads facing the epicardial surface of the right ventricle such as those over the lower mid-sternal region and the right upper abdomen have a negative main deflection. The left arm, the left upper abdomen and the left leg are found to be transition zones, i.e. their potentials vary greatly with the position of the heart. Goldberger maintained that a complete electrocardiographic technique should include /

/ include leads that record these five basic ventricular patterns as well as three basic auricular patterns which he also described; about twenty unipolar leads would be necessary from which a fairly detailed assessment of the electrical cardiac position could be made. He discounted the validity of Wilson's method of estimating cardiac position, as described above, since the extremities often face regions of the heart that the praecordial leads do not face and hence it may be impossible to correlate the two groups. Goldberger prefers to interpret any given praecordial or limb pattern in terms of his five basic ventricular forms, described above.

While therefore the ideal unipolar lead has not yet been found and variations in technique must continue and while the interpretation of any unipolar pattern rests on the hypothesis that there is a positive potential ahead of the activating impulse in the myocardium and a negative one behind it, the technique of unipolar electrocardiography has by this time come to rest on a foundation built by long practice and adequate mathematical proof. Side by side with the advances in the technical and theoretical fields summarised above, its contribution to the study of myocardial infarction has been explored by numerous workers in the past decade.

In the paper previously cited from the Michigan /

/ Michigan School (1944) Wilson et al, besides amplifying their interpretation of the normal praecordial unipolar electrocardiograms, summarised above, also extended their views on the significance of the patterns in myocardial infarction. They interpreted the central type of curve, viz. a deep QS deflection which is found where the whole thickness of the ventricular wall is dead as being due to the transmission to the praecordial electrode of the negative potential of the ventricular cavity through the inert "hole" or "window" provided by the infarct. In many infarcts, however, some fraction of muscle remains alive and gives rise to an embryonic R wave in the course of the QS deflection. It may only be a notch on the descending or ascending limb of the QS wave, but if the outer layers of the myocardium are still capable of responding to the impulse, the R wave is larger and, although still delayed, it reaches above the isoelectric level. Thus a QR complex is recorded. Such appearances are common at the margin of infarcts; here also it is usual to find sharp inversion of the T wave "due to an increase in the duration of the excited state at epicardial surface."

Goldberger (1945) also subscribes to the above views, He points out that if an infarct in the left ventricular wall is small both these leads which overlie its epicardial surface and those which overlie the /

/ overlie the unaffected regions of the ventricle may show a small normal q wave because both leads face the left side of the interventricular septum and record the spread of the stimulus from its left surface towards its central layers by a small initial negative deflection. However if the infarct is large a deep Q is recorded for the reasons stated by Wilson and summarised above. Goldberger finds that in leads which face the epicardial surface of the left ventricle the normal q wave is less than 0.04 sec. in duration whereas an abnormal Q wave or QS deflection measures more than 0.04 sec. in width and furthermore abnormal Q or QS waves are frequently associated with elevation of the RS-T junction and segment or with deep symmetrical T waves in the earlier stages of infarction. Such an association is uncommon with normal q waves although it can occur if a fair amount of muscle had survived the infarction. Nevertheless when the infarct heals RS-T changes tend to disappear although abnormal Q waves may remain indefinitely. In actual practice if a Q wave in a unipolar lead over the left ventricle constitutes the main ventricular deflection it is abnormal. Goldberger's interpretation of QR deflections is the same as Wilson's.

With regard to infarcts which involve the right ventricle Goldberger states that an abnormal Q may not appear because leads which overlie such an infarct are/

/ are also facing the right side of the septum and hence record a small normal r wave due to the spread of the stimulus from the left side of the septum towards its middle layers. However infarcts of the right ventricle near the apex may show abnormal Q or QS waves because the R wave normally recorded in this situation is due to the early arrival of the impulse at the cardiac apex and its passage through the right ventricular wall rather than to activation of the septum which is in any case in the same plane as the electrode and hence its electrical activities, being at right angles to this plane, are unlikely to be recorded by such an electrode.

Goldberger also studied the Q waves of unipolar left arm and left leg leads, pointing out that the presence of Q waves in these leads and hence in the standard limb leads is as much dependent on the position of the heart as on the situation and size of the infarction. He believes that the heart after an infarction occupies a different electrical position from that before infarction. In the first place a large area of muscle is no longer functioning and the force of contraction of the remaining muscle may cause abnormal torsion of the heart around any of its axes. In the second place changes in the size of the heart may occur after infarction due to decrease in output, cardiac decompensation or localised dilatation. In view of /

/ of these facts the differentiation of normal from abnormal Q waves in leads VL and VF is often difficult. As a result of his comparison of fifty cases of anterior infarction and one hundred uninfarcted controls either with or without ventricular hypertrophy Goldberger concluded that the Q wave in lead VL after infarction measures at least 50% of the amplitude of the succeeding R wave which is invariably present or 30% or more of the entire QRS and that it should be 0.04 sec. or more wide. As with abnormal Q waves in praecordial leads described above, the RS-T complex shows the usual signs of infarction but these are however temporary. It is emphasised however that such a QR deflection may occur in the left arm lead in the absence of infarction, e.g. if the left arm records potentials from the back of the heart and similarly infarction may be present when this pattern is absent. A deep QS wave in lead VL may be recorded if the left arm faces the central zone of the infarction; a vertical heart also causes this pattern in lead VL but in the latter case the P wave is inverted; in either case the T wave is inverted. Examples of the above circumstances occur in the text of the present work.

The abnormal Q of lead VL is transmitted to standard limb lead I. According to Goldberger, a Q wave in lead I is abnormal, if the accompanying r wave is small, if the Q is 0.04 sec. or more wide and if it is 1 mm. or /

/ or more deep but the diagnosis of anterior infarction is much less fallacious if it is based on the anterior praecordial leads and not on lead VL or standard limb lead I. However in the case of posterior infarction much reliance is placed on the left leg lead since, apart from oesophageal leads, it is the only one which faces the epicardial surface of the infarction. Based on fifty cases of posterior infarction and one hundred controls Goldberger's criteria for an abnormal Q wave indicative of infarction in lead VF are: the duration of an abnormal Q wave should be 0.04 sec. or more measured from its onset to return to base-line; its amplitude should be 60% of the succeeding R wave or 40% of the entire QRS complex. Such an abnormal Q in lead VF causes a similar wave in standard limb leads II and III; in lead III, according to Goldberger, a Q wave, to be abnormal, should last 0.04 sec. at least and should measure 50% or more of the tallest R in the standard leads, and, in lead II, it should be 25% or more of the size of the R wave of that lead.

The above criteria for an abnormal Q wave in lead VF were criticised by Myers et al (1949). They studied fifty cases in which the final decision regarding the presence or absence of posterior infarction was based on the findings in oesophageal leads. Forty-five of the fifty cases had fulfilled Pardee's criteria for an /

/ an abnormal Q wave in standard limb lead III but only twenty-four of the forty-five gave positive signs of infarction in the oesophageal leads; in the remaining twenty-one posterior infarction could be excluded. As Myers points out, hearts which are horizontal, semihorizontal or intermediate have a tall R wave and upright T wave in lead VL derived from the left ventricle but because the galvanometric connection to the left arm in lead III is the reverse of that in lead VL, a deep QS and inverted T are recorded in lead III. However, a Q wave in lead VF is frequently an indication of posterior infarction. Twenty-two of the twenty-four subjects with proved posterior infarction showed it but only three of the twenty-one subjects with a Pardee Q3 and negative oesophageal leads. Thus lead VF was a more accurate basis for diagnosis of posterior infarction than the Pardee criteria for standard limb lead III. On these grounds Myers set down criteria for the diagnosis of an abnormal Q wave in lead III somewhat different from those of Goldberger, viz. (i) the voltage of the QRS complex of lead VF should be at least 0.5 millivolt; (ii) the duration of the Q wave should be 0.04 sec. Myers measured from the onset of the Q wave to its nadir while Goldberger measured from onset to return to baseline; (iii) the Q wave should have an amplitude of 25% or more of that of the succeeding R wave. If Goldberger's /

/ Goldberger's criteria had been applied to Myers' forty-five cases, two of the twenty-one uninfarcted controls would have been considered abnormal, whereas four cases with known infarctions had Q/R ratios not considered abnormal by his standards and two others were borderline in his view. In the present work Myers' criteria for an abnormal Q wave in lead VF have been adopted.

Wilson's explanation of the coronary QS wave, viz. that it represents the unaltered transmission of the negative potentials of the ventricular cavity through the "hole" or "window" of transmural infarcted tissue has recently been subjected to experimental investigation by Prinzmetal et al (1953). He used a needle-like silver plunge electrode insulated except for its tip which he inserted by stages through the ventricular wall of the dog, taking electrocardiograms at each step. He found positive potentials only in a shell of epicardial muscle; muscle placed more deeply was negative throughout the process of depolarisation and gave QS deflections without R waves. Similarly the septum was positive on its right side, the positivity increasing as the electrode was drawn from the surface of the right side through its substance towards its middle layers; thereafter as it transversed the left side of the septum, the potentials became less positive and finally became negative as the /

/ the surface of the left side was approached. Negative potentials were registered in the ventricular cavity. Prinzmetal labelled the QS deflections found intramurally "QSm" in contrast to those found in the cavity "QSc" and he himself is able to distinguish them in his experimental curves but he does not consider that the usual clinical procedures will allow of such differentation. He next applied his plunge electrode to experimentally produced infarcts. He obtained QSc curves throughout the thickness of infarcts which were entirely fibrous, thus confirming the work of Wilson. When he investigated infarcts with surviving muscle he found positive potentials in the epicardial layers only; thereafter they were negative throughout the remainder of the thickness of the wall. Thus, he postulated, a QS deflection found over an infarct may be due to cavity potentials, to intramural potentials or to a mixture of both - the more surviving muscle the greater the intramural negativity. He also investigated the coronary QR wave, which, according to Wilson, represents the transmission of the negative cavity potentials to the epicardium with a resultant initial downward deflection, after which overlying intact muscle contributes a positive potential, represented by the late R deflection. He was unable to record Q waves over his experimental infarctions or over the epicardium /

/epicardium of regions of which the endocardium had been cauterised. He considers that some degree of epicardial damage is necessary for the production of a QR wave. He concludes therefore that epicardial or praecordial electrocardiograms provide a fair representation of epicardial potentials only, independently of the state of the underlying intramural muscle. In the present work, the concepts of Wilson have been retained.

Similarly, the displacement of the RS-T segment has been investigated by experiment by several workers. Pruitt and Valencia (1948) produced myocardial infarction in dogs by coronary ligation and found that there was upward displacement of the RS-T segment in leads from a portion of the ventricular cavity adjacent to the infarct as well as in curves obtained directly over the infarction. They believed that the electromotive forces responsible for upward RS-T displacement on both aspects of these transmural lesions were due to the boundaries between injured and uninjured muscle at the peripheral margins of the lesion. Hellerstein and Katz (1948) produced focal subepicardial lesions of the free walls of the ventricles and subendocardial lesions of the free walls and of the septum by physico-chemical means. Their results were similar to those of Pruitt and Valencia in so far as they found positive displacement

/ displacement of the RS-T segment when the electrode was placed on a subepicardial infarct and also when the electrode was within the ventricular cavity in the neighbourhood of a subendocardial infarct but they made further significant observations. In both cases there is negative RS-T displacement on the other side of the affected portion of the ventricular wall; in both cases the cavity potentials of the RS-T period are transmitted unchanged through the sound myocardium of the wall opposite the infarct; i.e. if there is a subepicardial infarct of the anterior wall, an electrode on the epicardium of the posterior wall registers RS-T depression; if the infarct of the anterior wall is subendocardial, the electrode in the same position records a positive RS-T displacement. They also found that at, or just beyond, the periphery of subepicardial lesions, the displacement of the RS-T segment fell to zero from its positive level over the infarct. More distally still, it showed a zone of negative displacement, beyond which it again become isoelectric. By this time the electrode was approaching the diametrically opposite wall where typical RS-T depression was found.

While the latter findings await confirmation and clinical application, the broader generalisations regarding RS-T displacement have been adopted in the /

/ the present work; viz. that immediately over the subepicardial infarct, there is positive RS-T displacement and that over the external surface of a portion of ventricular wall of which the subendocardial layers are infarcted there is RS-T depression. Lastly, when the infarct is of some size there is RS-T depression in leads which face the cavities of the ventricles and in leads over the unaffected wall opposite the infarction. To align this statement with modern theory, the premise is necessary that the subendocardial layers have escaped infarction.

In the paper previously cited, Wilson (1944) extended the application of unipolar electrocardiography to problems of situation and size of infarction in the solution of which he had predicted the value of unipolar leads as early as 1934. On the basis of praecordial electrocardiograms he subdivided anterior infarctions into those involving chiefly the anteroseptal wall of the left ventricle and those involving the anterolateral wall. The latter frequently includes the former. An infarct is considered to be anteroseptal in situation if diagnostic changes are restricted to praecordial leads V2 and V3. In such cases the leads over the left side of the praecordium show few, if any, abnormalities, and if these hearts are in the semihorizontal or horizontal position, the pattern of these leads is transmitted to /

/ the left arm and thence to standard limb lead I, which is therefore similarly uninformative of the presence of the anteroseptal infarction.

An infarct is said to be anterolateral if diagnostic signs are present in leads V4 and V5; they may either be confined to these leads or all four leads, V2 to V5 inclusive, may show the pattern of infarction, marginal or central. In anterolateral infarctions diagnostic signs are frequently seen in lead VL and hence also in standard limb lead I. While QS or QR deflections are the classical signs of infarction, as previously discussed, Wilson attached great weight to another pattern, viz. progressive diminution in size of the R wave as the electrode passes from the V1 to the V4 position, thus indicating increasing inability of the underlying muscle to respond to the excitatory process. He considered that this sign indicated infarction of the anterior wall of the left ventricle.

Similarly, the large group of posterior infarctions are divided into "plain" posterior and posterolateral. An infarct is said to be plain posterior in situation if diagnostic signs are found in lead VF but not in any praecordial leads, the anterior of which usually show the signs reciprocal to infarction. If, in addition to lead VF, the axillary leads show signs of infarction, usually a marginal pattern, then the infarct is considered

/ considered to be posterolateral in situation.

Both types of posterior infarction show diagnostic signs in oesophageal leads. The diagnostic possibilities of these leads were explored by Nyboer (1941). At the ventricular levels of the oesophagus the QRS complexes and T waves are chiefly upright. This is in contrast with their form at auricular levels where the QRS complexes show an initial downward deflection or are chiefly negative and the T waves are inverted. When the posterior ventricular wall is infarcted however, deep Q waves occur at the ventricular level with elevation of the RS-T segment, and, later, inversion of the T wave. Such a tracing may be differentiated from a normal tracing taken at the auricular levels by the distinctive form of the P wave in the latter - it shows an intrinsic deflection.

Sometimes a plain posterior infarction as indicated by lead VF has also typical signs of infarction in the ensiform lead which Wilson took as a routine procedure. He considered that this indicated involvement of the inferior and basal regions of the posterior wall. In such cases praecordial leads V1 and V2 do not show the reciprocal signs of infarction commonly found in posterior infarction, on the contrary the R wave may be absent or very small in these leads.

Combined patterns, viz. T1 and T3 types, may be

/ be explained by reference to praecordial and unipolar limb leads. If an infarct is posterolateral in situation and if the T abnormalities of the axillary leads are transmitted to the left arm, standard limb lead I will probably display them also. Hence there are classical signs of posterior infarction in standard limb leads II and III combined with T abnormalities in standard limb lead I. A similar grouping of signs may be found if a small anterior infarction complicates a pre-existing posterior infarction, and, lastly, Wilson considered that it may also arise in certain anteroseptal infarctions where "because of some peculiarity in position of the heart, the potential variations of the infarcted area are transmitted to the left leg."

Wilson again expanded his views on the diagnosis of myocardial infarction in 1946 at the end of a discussion on the theoretical aspects of unipolar electrocardiography. He described six cases each of which presents an unusual or unexplained feature. He himself was well aware of the existence of these problems and of the locality to which further investigation should be directed. The first case was an anteroseptal infarction diagnosed from leads V1, V2 and V3. The apical and axillary leads were normal but lead VL had terminal inversion of the T wave and standard limb lead I had a flat T wave. The derivation of the

/ the left arm potentials is not clear. The second case was a high lateral infarction, proved by multiple leads at levels of the 4th and 3rd left intercostal spaces but the right praecordial leads did not show high R and T waves as they usually do in high lateral infarction. (Earlier in the same year, Wilson had published six cases of lateral infarction including the case above quoted. He considered it might be a high anterolateral infarction.) The next case showed the typical signs of a posterolateral infarction; at postmortem examination, the posterior region of the left ventricular wall showed a recent infarct whereas the lateral wall showed old scarring. The fourth case exemplified the need for serial tracings - a 12 lead electrocardiogram was practically normal eight days after the onset of praecordial pain which recurred daily. Two and a half weeks after onset, signs of a high lateral infarct were present. The fifth case showed the signs of right bundle branch block and posterior infarction in the unipolar limb leads and those of anteroseptal infarction in the praecordial leads. At autopsy the infarction extended in a crescent from the anteroseptal wall, through the central layers of the septum to the posteroseptal wall; in addition there was old scarring of the posterolateral wall. The last case presented the enigma of classical signs of old posterior infarction in standard limb leads II and III as well as in lead VF and

/ and in multiple oesophageal leads but, many years previously, the standard limb leads had been taken in a physiological experiment and they presented the same signs. At no time did the subject experience symptoms likely to be attributable to myocardial infarction.

It was clearly necessary that a close comparison be made between the diagnosis of site and age of infarction, reached by full unipolar electrocardiography and the postmortem findings - a need exemplified by the two cases described above. This gap was bridged by the work of Myers et al, published in 1948 and 1949. They performed 161 postmortem examinations on subjects of whom they had at least one set of multiple electrocardiograms. They divided their cases into, i) anteroseptal; ii) large anterolateral; iii) posterior; iv) posterolateral; v) anteroposterior; vi) lateral infarction, and vii) infarctions of interventricular septum and right ventricle. Their interpretation of electrocardiographic patterns followed the Wilson school but they were more sensitive to the effect of cardiac position both as a cause of the vagaries of pattern upon which that due to infarction is superimposed and as a reason for failure to localise an infarct antemortem. Their findings are reviewed in the appropriate sections of the present work in so far as they are apposite to it.

However, electrocardiographic and postmortem /

/ postmortem correlations do not solve the problems encountered in clinical cases. The present work was undertaken with a view to reassessing the assistance which might be expected from standard limb leads on the one hand and unipolar leads on the other in the diagnosis of infarction and from the latter in the estimation of the situation, size and healing of infarctions. The probability has been kept in view that a study of the retrogressive changes in infarction recorded in electrocardiograms would have a bearing on the clinical prognosis.

SECTION III

ANTEROSEPTAL INFARCTION

SECTION III

ANTEROSEPTAL INFARCTION

INTRODUCTION

It is now customary to make a diagnosis of infarction of the anteroseptal wall of the left ventricle if a QS deflection or an abnormal QR deflection is found in leads V2 and V3 or in leads V3 and V4 or in all three leads. While a QS deflection in these leads is always abnormal, a QR deflection is not necessarily so. Goldberger (1945) states that in leads which face the epicardial surface of the left ventricle, which is the usual relationship for leads V3 and V4, although not for V2, a Q wave is abnormal when it measures more than 0.04 sec. from onset to return to baseline or when it forms the main ventricular deflection. Myers (1948) considers it abnormal if it measures 0.02 sec. or more from onset to nadir and if its amplitude is 25% of that of the succeeding R wave. All fifteen cases of anteroseptal infarction discussed in this work have QS deflections so that doubts with respect to the Q/R ratio do not arise. The classical electrocardiographic pattern of anteroseptal infarction should have a normal lead V1, i.e. the small initial upright deflection or R wave of lead V1 should persist. Being derived from the

/ the normal activation of the left side of the septum its persistence indicates intact septal function, at least in its anterior part. Sometimes the R wave of lead V2 also survives but it is smaller than that of lead V1 since it is nearing the site of infarction. This decrease in the size of the R wave as the electrode passes from the V1 to the V2 or even to the V3 positions was considered by Wilson (1944) to be an important sign of anteroseptal infarction. Similarly, any Q waves registered by leads V5 and V6 or by lead VL should be small and of brief duration since they are derived from the same source as the R wave of lead V1, viz. from early activation of the left side of the septum.

While this QRS pattern is typical of anteroseptal infarction Myers (1948) points out that it may also occur in lateral infarction and in right ventricular dilatation. In the former, if severe counter-clockwise rotation of the heart about its own long axis is present, then the potential variations of the lateral ventricular wall may be referred to the praecordium and abnormal QR deflections may be recorded by leads V2, V3 and V4. In right ventricular dilatation and hypertrophy lead V1 may show a normal RS deflection but in leads further to the left, there may be reduction or disappearance of the R wave.

Modifications of the typical pattern are common in anteroseptal infarction and have been discussed by /

/ by Myers (1948), whose views may be summarised as follows. Frequently lead V1 does not register a small initial R wave but, like V2, shows only a deep QS deflection. In view of the septal origin of the R wave of lead V1 its disappearance indicates involvement of part of the septum, in particular of its anterior portion so that the negative potentials of the left ventricular cavity are transmitted through the septal infarct to the right praecordium and exceed any positive potentials derived from activation of septal remnants together with those from the free wall of the right ventricle. While therefore abnormal QS deflections in leads V1 and V2 tend to occur as a result of extension of an anteroseptal infarction into the septum they are also found in primary septal infarction, which is, however, an uncommon lesion. Proof that such QS deflections are due to infarction is afforded by the demonstration of a small initial R wave derived from the intact posterior part of the septum in a lead further to the right than the V1 position, say lead V3R. Similarly if leads further to the left, viz. leads V3 and V4 show abnormal Q waves typical of a marginal zone of infarction, this constitutes indirect evidence that the QS pattern of leads V1 and V2 is due to infarction. During the acute stage, the /

/ the RS-T segment of leads V1 and V2 may provide additional diagnostic signs, viz. elevation of the RS-T junction and straightening or upward convexity of the RS-T segment. If the above ancillary evidence is not available then QS deflections in leads V1 and V2 cannot be attributed unequivocably to infarction since they may occur occasionally in normal horizontal hearts and also in left ventricular hypertrophy if by backward rotation of the apex, the mitral orifice is tilted forwards and towards the right so that the negative potentials of the left ventricular cavity are transmitted to the electrode in the V1 and V2 positions and are large enough to exceed the positive potentials arising from the septum and the free wall of the right ventricle. The same pattern also occurs in left bundle branch block since in this condition the septum is activated entirely from its right side and thus an initial downward deflection is registered in leads V1 and V2.

The RS-T pattern over an anteroseptal infarction depends largely upon its age although it must be admitted that a series of electrocardiograms displaying sequential RS-T changes is much more reliable as an indication of the age of an infarct than is one tracing. Early in the stage of injury the RS-T junction is elevated 2-8 mm. above the iso-electric line and from /

/ from this point the RS-T segment rises in a straight line or in a line with upward convexity to the T wave which is monophasic and upright. These changes may precede histological evidence of infarction, a fact which is in accord with experimental work in animals (Blumgart et al, 1941). The RS-T displacement soon begins to recede and the terminal portion of the T wave begins to dip below the isoelectric line. The T wave ultimately becomes inverted and cove-plane in contour. After reaching maximal depth it gradually recedes while the RS-T junction approaches the isoelectric line and the RS-T segment becomes less dome-shaped. Eventually the T wave becomes flat or upright although fixity of the RS-T pattern may occur at any point. Frequently the characteristic cove-plane inversion of the T wave may become permanent and even upward displacement of the RS-T segment may persist in occasional instances. The above changes in combination with the QRS pattern previously described are pathognomonic of anteroseptal infarction when they occur in leads V2, V3 and/or V4 but the above RS-T changes, occurring alone, are not necessarily indicative of infarction because similar changes occur in acute right ventricular dilatation and in acute pericarditis.

In acute anteroseptal infarction sharply inverted T waves may be recorded by leads V5 and V6 which /

/ which frequently overlie the zone of ischaemia around the infarction. There is no preceding displacement of the RS-T segment and there are no significant QRS changes. From these leads, the T inversion is transmitted to the left arm and thence to standard limb lead I. In view of the reversibility of ischaemia it is not surprising that these T waves usually return to normal or revert to a previous pattern of left ventricular hypertrophy or, in the course of a year or so, develop the signs of left ventricular hypertrophy.

The present section of this work deals with fifteen cases of anteroseptal infarction, of which the following are the case reports and electrocardiograms.

Case 1, J.Y. male, 51 years.

The first 12-lead electrocardiogram was obtained one week after onset of illness. The standard limb leads are similar to those taken four hours after onset but unfortunately on that occasion unipolar leads were not taken. Standard limb lead I shows a small Q wave in both instances measuring 1 mm. but whereas in the first electrocardiogram it is one sixth of the amplitude of the R wave, in the second it is one quarter. Examination of the unipolar praecordial leads shows that the Q wave of standard limb lead I is the summation of the abnormal Q wave of lead VL and of the reverse of the normal initial positive wave of lead VR which is derived either from the anterior cardiac wall or from the interventricular septum. Hence the Q wave of standard limb lead I is only partly derived from pathological signs. Probably of greater significance therefore is the decrease in the height of the R wave and the slight decrease in size of the positive T wave. The pattern in lead VL is clearly derived from the marginal zone of infarction, i.e. there is a Q wave which is almost half of the size of the R wave although both are small; there is slight elevation of the ST segment and the T wave is flat. The tall R wave of lead VF indicates vertical or semivertical cardiac position.

/ position. The praecordial leads show absence of the normal small initial upright deflection in leads V1 and V2. This sign almost certainly indicates septal infarction in this case since it is accompanied by elevation of the ST junction in these leads and by pathognomonic signs of infarction in leads V3 and V4. Furthermore the heart is in a vertical or semivertical position, not a horizontal one, and it is only in the latter position that cavity potentials may be referred to the right praecordium and hence there may be no small initial R wave in leads V1 and V2 normally. Leads V3 and V4 show the classical signs of infarction of the anteroseptal wall, central in lead V3 and marginal in lead V4.

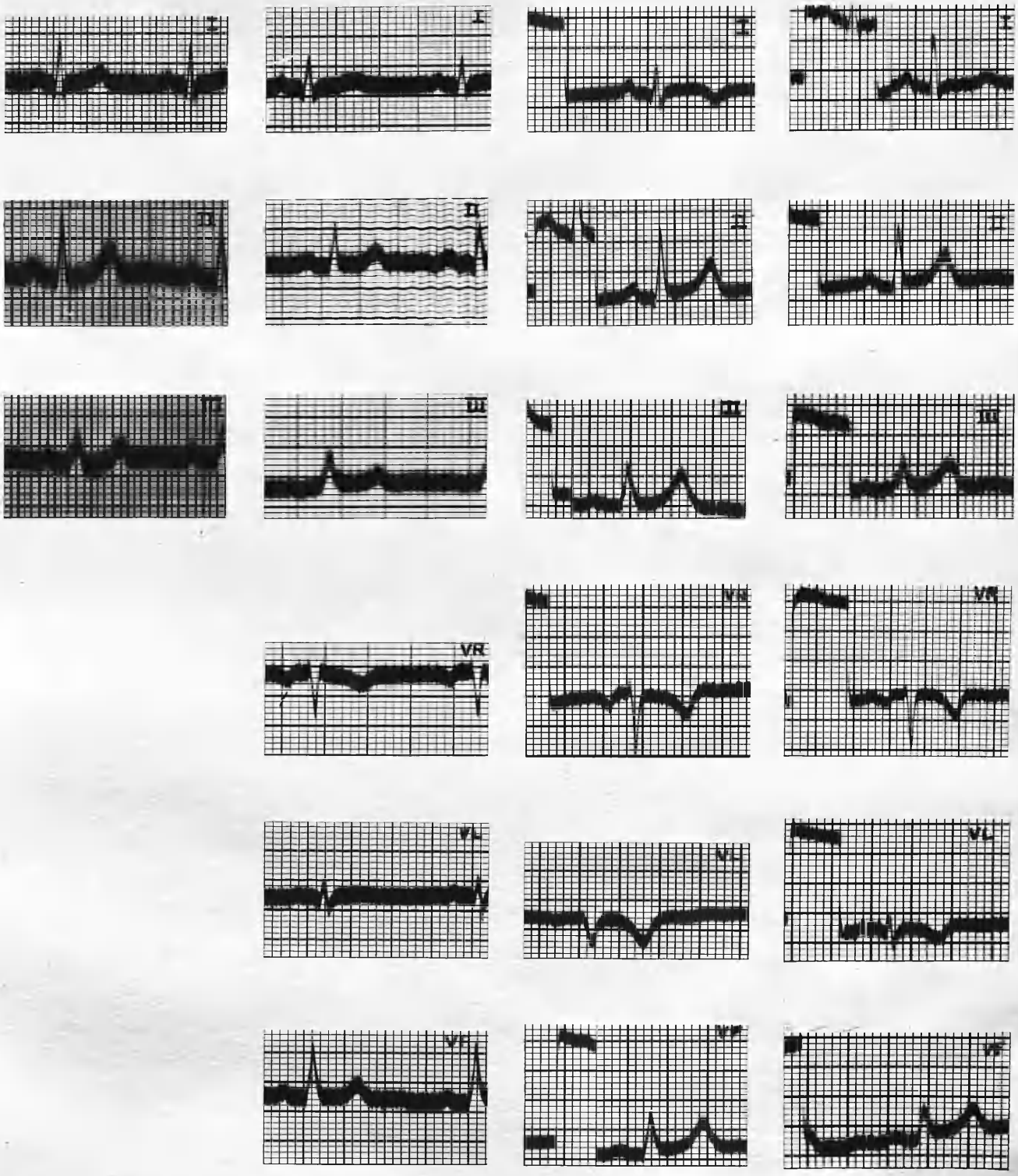
During the following week, the infarction spread to involve the lateral wall. Lead VL now reflects the central zone of infarction, a transmission facilitated by forward rotation of the apex or clockwise rotation of the heart around its own long axis both of which are common in vertical hearts.

In spite of such a pathognomonic figure in lead VL standard limb lead I shows little change in its QR ratio because, as before, the signs in lead VL are frankly mitigated by the reciprocal of lead VR. The S wave of standard limb lead I has become more prominent representing, as it does, the end of the QS deflection of

/ of lead VL. The ST-T formation of standard limb now shows an ST plateau and inversion of the T wave. The praecordial leads show that the infarct has spread to the lateral wall and that the extension is laminar. There is conspicuous deepening of the Q wave and shortening of the R wave of lead V4. As a corollary of the shortened R wave, the S wave is also deeper being derived from intact ventricular muscle elsewhere, which now forms a proportionately larger amount of the muscle mass than in the previous tracing. The marginal zone is now shifted to the V5 level. Concurrently the ST-T deflections are evolving in the usual manner. Very deep cove-plane T waves are seen in leads V3 and V4, measuring fifteen and twelve millimetres respectively.

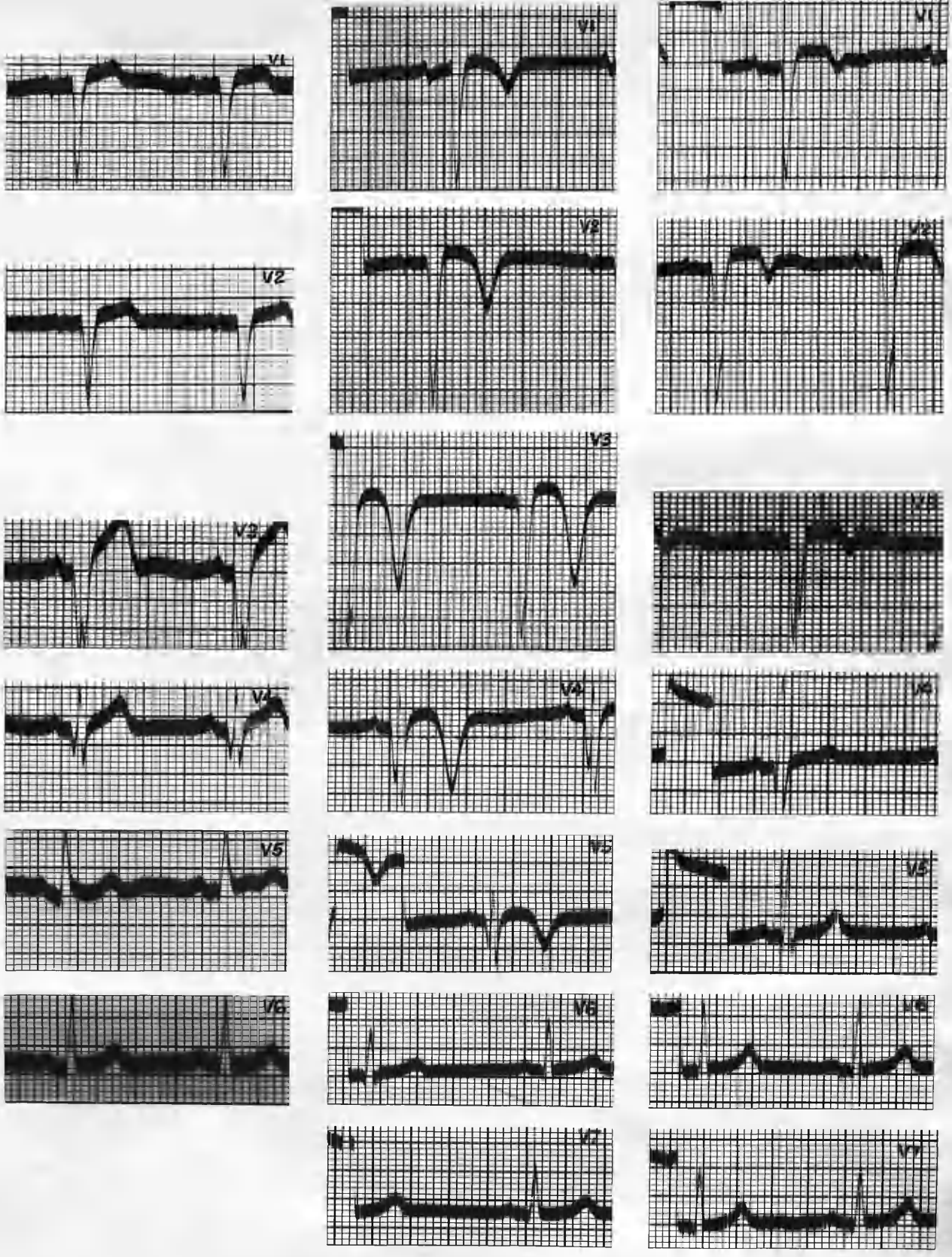
By seven and a half months after onset the standard limb leads are again within normal limits. Lead VL shows a small R wave again, the pattern reverting to a marginal type. The praecordial leads show a striking recession of signs in leads V5 and V4 both of which are again within normal limits. Lead V3 now shows severe splintering of its deep QS deflection, the positive element rising just sufficiently above the isoelectric level to be called an R wave. The same component is represented by notching of the deep QS deflection of lead V2. In lead V1, the QS complex remains unaltered.

/ There is still elevation of the ST junction in leads V1, V2 and V3 and persistent inversion of the T waves. Thus there has been very good recovery at the V5 and V4 levels and at least laminar recovery at the V3 level.



CASE 1, J.Y., MALE, 51 YEARS.

4 HOURS AFTER ONSET.	1 WEEK AFTER ONSET.	2 WEEKS AFTER ONSET.	7½ MONTHS AFTER ONSET.



CASE 1, J.Y., MALE, 51 YEARS.

1 WEEK AFTER ONSET.

2 WEEKS AFTER ONSET.

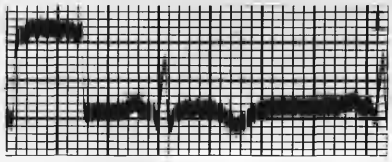
7½ MONTHS AFTER ONSET.

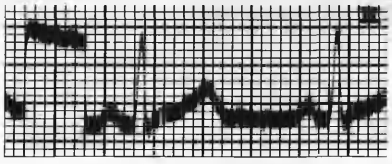
Case 2, A.J.M., male, 40 years.

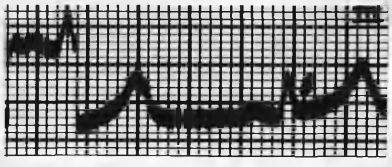
This patient, an acid-worker, had had severe praecordial pain lasting for three hours about a week before admission to hospital. He had had no previous chest pain. General condition was satisfactory. The apex-beat was diffuse and the cardiac sounds were "soft and tic-tac" in character. Blood pressure was 128/72.

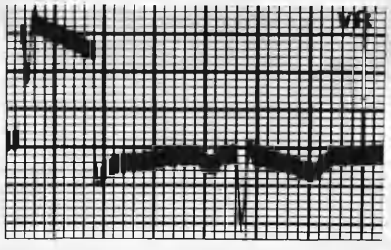
Electrocardiograms were taken ten days after onset. The standard limb leads are very suggestive of an anteroseptal infarction with upward convexity of the ST segment and sharp inversion of the T wave in lead I. The praecordial leads show a small R wave in lead V1 diminishing practically to nil in lead V2 and a deep QS deflection in lead V3. This is a typical pattern of anteroseptal infarction. Cove-plane T waves are seen in leads V3 and V4 where they measure seven and six millimetres respectively but the early phase of the ventricular complex is of normal transitional RS form, indicating more or less intact left ventricular muscle in this region. It is clear that lead VL has derived its potentials from the central zone of infarction for it presents a broad notched QS deflection. It is probable therefore that the cardiac apex was rotated forwards so that the central zone faced the left arm while intact left ventricular wall at a lower level was referred to the /

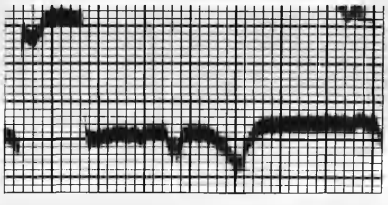
/ the apical and axillary leads. Such a transmission of potentials would also be favoured by the vertical position of the heart which is indicated by the tall R in lead VF and especially if clockwise rotation about its own long axis were added. The pattern presented by lead VL is greatly modified by the reciprocal of the normal lead VR in the formation of standard limb lead I.

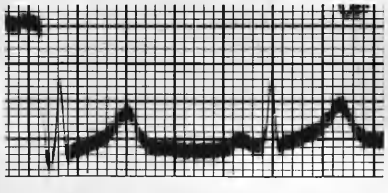


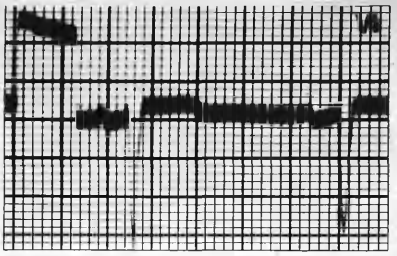


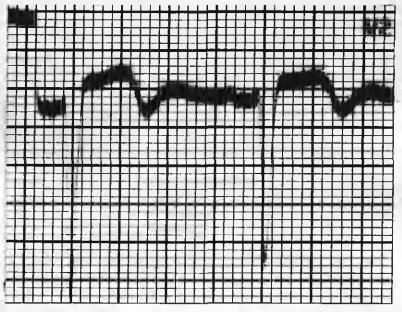


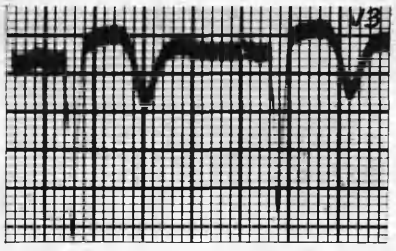


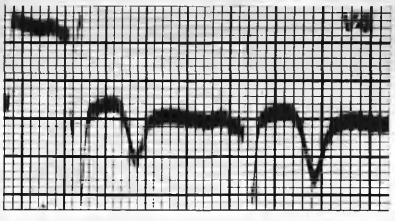


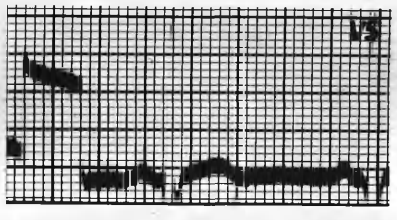


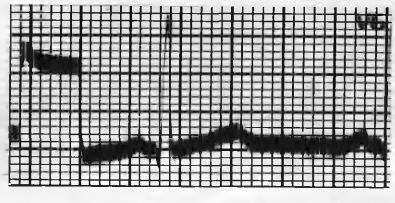


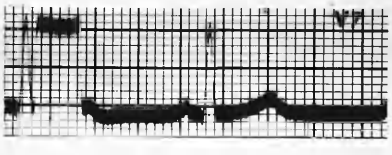












CASE 2, A.J.M., MALE, 40 YEARS.

10 DAYS
AFTER ONSET.

10 DAYS
AFTER ONSET.

Case 3, J.M., male, 57 years.

This patient, a shop-keeper by occupation, complained of praecordial and substernal pain of several hours duration occurring six days previous to his attendance at the out-patient department. The pain had not recurred and although the patient had remained off work, he did not feel ill.

General condition was satisfactory. There was no abnormal physical sign on cardiac examination. Blood pressure was 130/80.

He was advised to rest in bed at home. He had no further pain and was able to return to work.

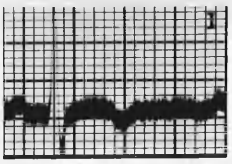
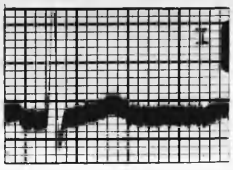
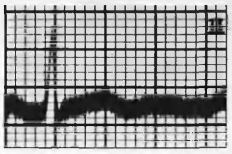
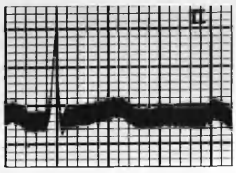
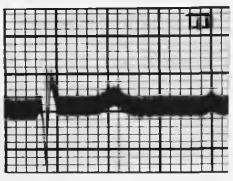
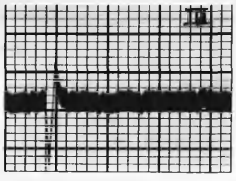
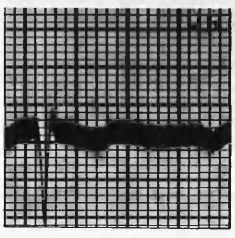
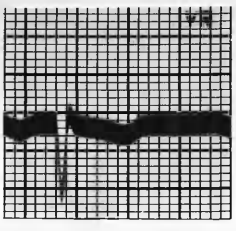
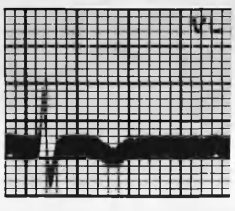
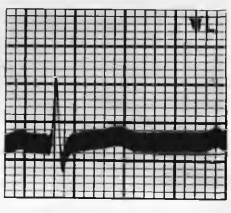
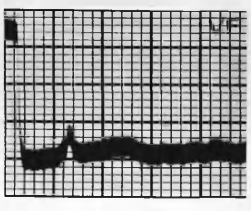
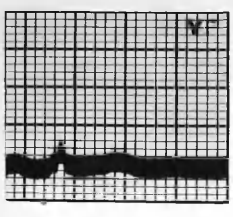
An electrocardiogram obtained six days after onset shows upward convexity of the ST segment of lead I without however elevation of the ST junction. There is also inversion of the T wave in lead I. Left axial deviation is present. The unipolar limb leads indicate that the electrical position is semihorizontal; the deflections of lead VF are small whereas those of lead VL are fairly large and are closely similar to those of standard limb lead I. The unipolar praecordial leads show a splintered QS deflection in leads V1 and V2. In view of the horizontal electrical position such a pattern may be a normal variant and not an indication of septal infarction /

/ infarction but the reappearance of an early small R wave in these leads one month after onset of illness indicates that its early absence is a sign of septal involvement. In lead V3 in the early tracing there is a significant Q wave followed by a shortened R wave indicating that this lead is over the marginal zone of infarction. This infarct would therefore appear to involve the anterior part of the septum and the adjacent anteroseptal wall. In spite of the limitation of QRS changes to the first three praecordial leads there are ST-T changes in all six praecordial leads, viz. straightening or upward convexity of the ST segment and inversion of the T wave. The deepest cove-plane inversion of the T wave is seen in lead V3. One month after onset, in addition to the reappearance of the early R wave in leads V1 and V2 as previously mentioned, there is increase in the later R wave in lead V3 already forecast in lead V2 by the notching, already noted, occurring earlier in the ventricular deflection. At the same time the Q wave of lead V3 has diminished considerably.

At this level, viz. over the free wall of the left ventricle close to the anterior terminus of the septum there has been a fair return of function. ST-T changes are now practically confined to the first four /

/ four praecordial leads and chiefly consist in sharp inversion of the T waves. Lead VL has returned to normal and hence also standard limb lead I.

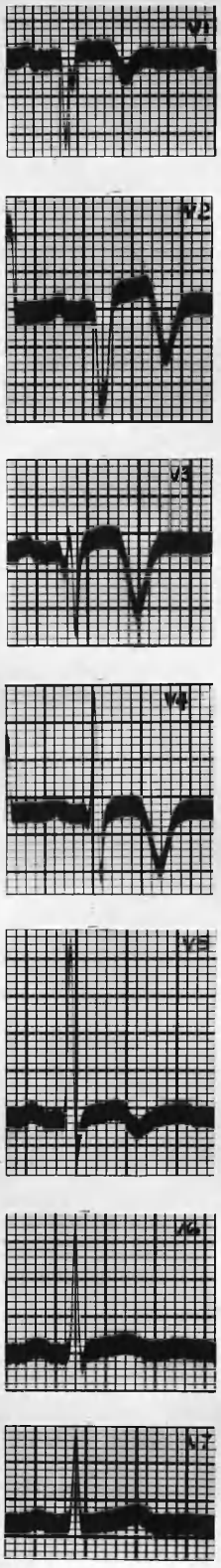
This case demonstrates fairly rapid healing in a septal and anteroseptal infarct in a horizontal heart.

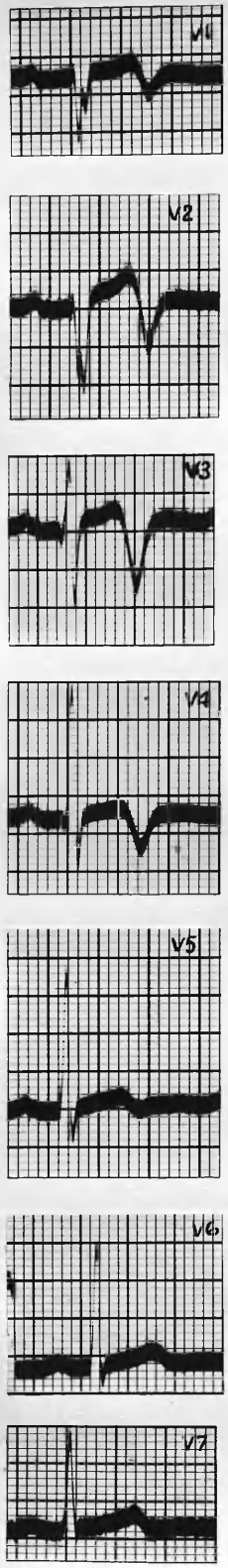













CASE 3, J.M. MALE, 57 YEARS.

6 DAYS
AFTER ONSET.

1 MONTH
AFTER ONSET.





CASE 3, J.M. MALE, 57 YEARS.

6 DAYS
AFTER ONSET.

1 MONTH
AFTER ONSET.

Case 4, J.C. male, 53 years.

This patient was admitted a fortnight after the onset of praecordial pain. On the evening before admission collapse with loss of consciousness supervened. On recovery of consciousness, praecordial pain was very severe. On admission blood pressure was 90/62; the pulse was thready and the cardiac sounds were very soft. In the next few days blood pressure rose to 110/70 at which level it remained throughout his hospital stay.

A 13 - lead electrocardiogram taken six days after onset shows the typical signs of an anterior infarction in the standard limb leads. The significance of the tiny Q wave in lead I is heightened by the shortness of the R wave which follows it. There is elevation of the ST segment in lead I and depression in leads II and III. The pattern of standard limb lead I is clearly derived from that of lead VL with little modification from lead VR the deflections of which are small. Lead VL has derived its potentials from the marginal zone of infarction which would indicate that the infarct has encroached on the lateral wall of the left ventricle and therefore that the infarct is in fact anterolateral and not limited to the anteroseptal wall. However the failure of leads V5 or V6 to show Q waves precludes this case from the

/ the anterolateral group, although no rigid distinction can be made between the two types. The praecordial leads show the typical signs of an anteroseptal infarction; deep QS deflections are seen in leads V1 to V4 inclusive and upward convexity of the ST segment is seen in leads V3, V4 and V5.

The association of a persistent R wave with upward convexity of the ST segment in lead V5 would suggest pericarditis or subepicardial involvement at this site.

By seven weeks after onset there is conspicuous left axial deviation in the standard limb leads with broadening of the QRS deflections and shallow inversion of T1. The unipolar leads indicate that the axis of the heart is now semihorizontal or horizontal. It is probable that this axial deviation is due to some recovery of left ventricular activity and not to an anatomical shift of axis in so far as there is a considerable increase in height of the R wave in lead V5 and in lead V4 it has emerged for the first time, indicating some return of left ventricular function. In addition lead V4 shows a further positive wave causing notching in the latter part of the QRS deflection; this probably indicates resuscitation of islets of muscle within the substance of the left ventricular wall.

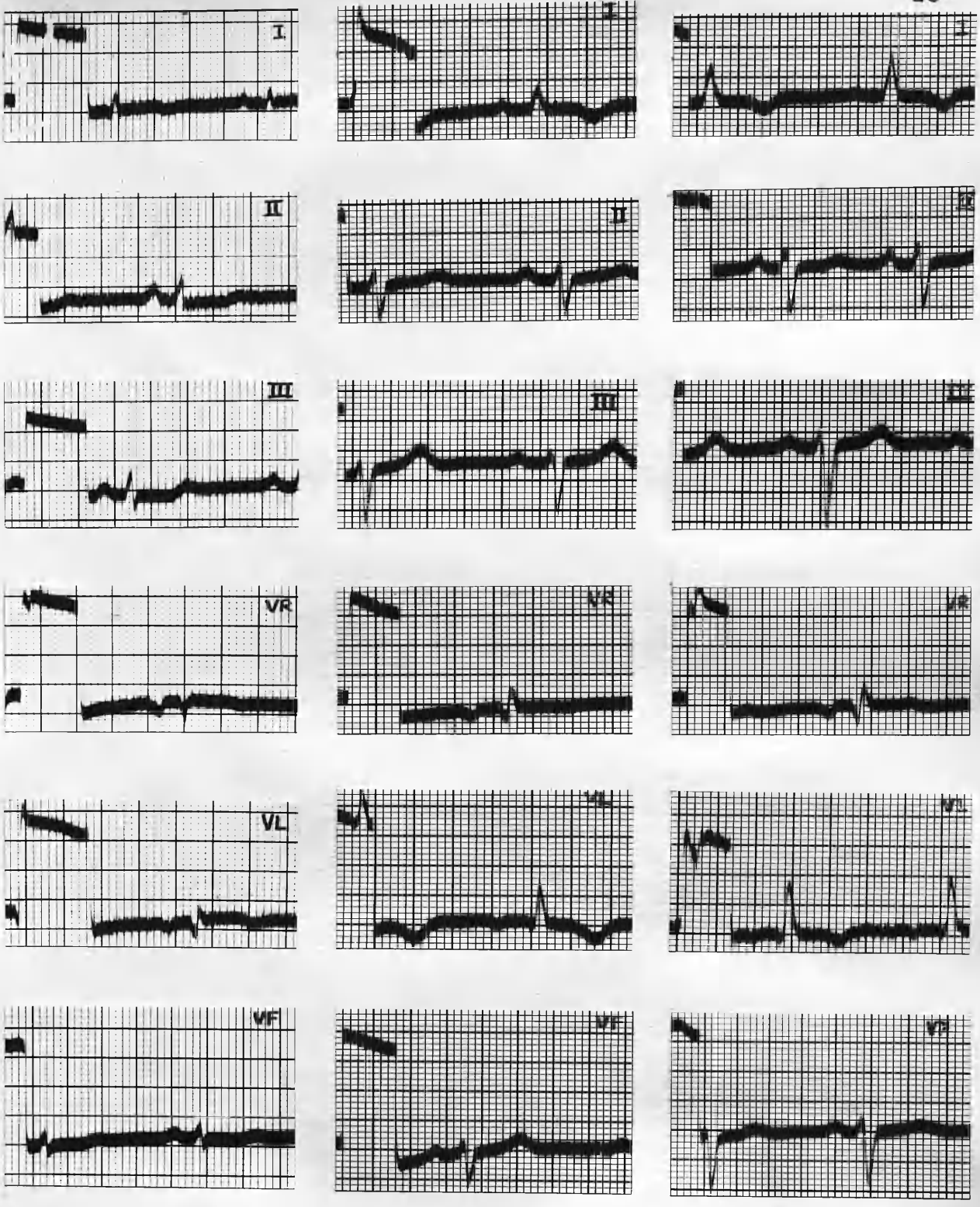
The patient was seen again eleven months after onset of illness. He complained of slight praecordial pain

/ pain if he walked more than two hundred yards. Blood pressure was 100/60. The chest was rather short and deep. The apex-beat was faintly palpable. The first cardiac sounds were soft. The patient was working steadily as a hairdresser by this time.

The final electrocardiographic pattern shows frank left axial deviation with broadening of the QRS deflection in the standard limb leads. In association with the inversion of the T wave in lead I the possibility of early left bundle branch block would arise from consideration of the standard limb leads alone but it is not confirmed from the praecordial leads. Lead VR now shows a pattern commonly derived from the back of the heart a derivation facilitated by backward rotation of the apex of a horizontal heart. Lead VL shows a short blunt Q wave followed by a slightly delayed R wave indicating local conduction defect in the lateral wall of the left ventricle. The praecordial leads show further signs of repair. The R wave has increased in height in leads V7, V6 and V5. In lead V4, both the early R wave and the late positive notching of the S wave are more conspicuous and an early R wave has now emerged in lead V3 indicating some return of function in subendocardial muscle. A minute R wave can be detected in lead V1 suggesting some recovery in septal function. However inverted or diphasic T waves persist in some measure throughout the

/ the praecordial leads, the greatest degree of inversion being seen in lead V4.

This case represents several features commonly found in partially healed infarcts: (i) the heart is horizontal with backward rotation of the apex. This position is frequent in healing anterolateral infarctions, and there is little doubt that the upper lateral wall was involved to some extent in the present case; (ii) there is a conduction defect in the lateral ventricular wall; (iii) there is fairly active recovery at the leftward margin of the infarct; (iv) there is persistent deformity of the T waves as an indication of myocardial ischaemia or dilatation in general.

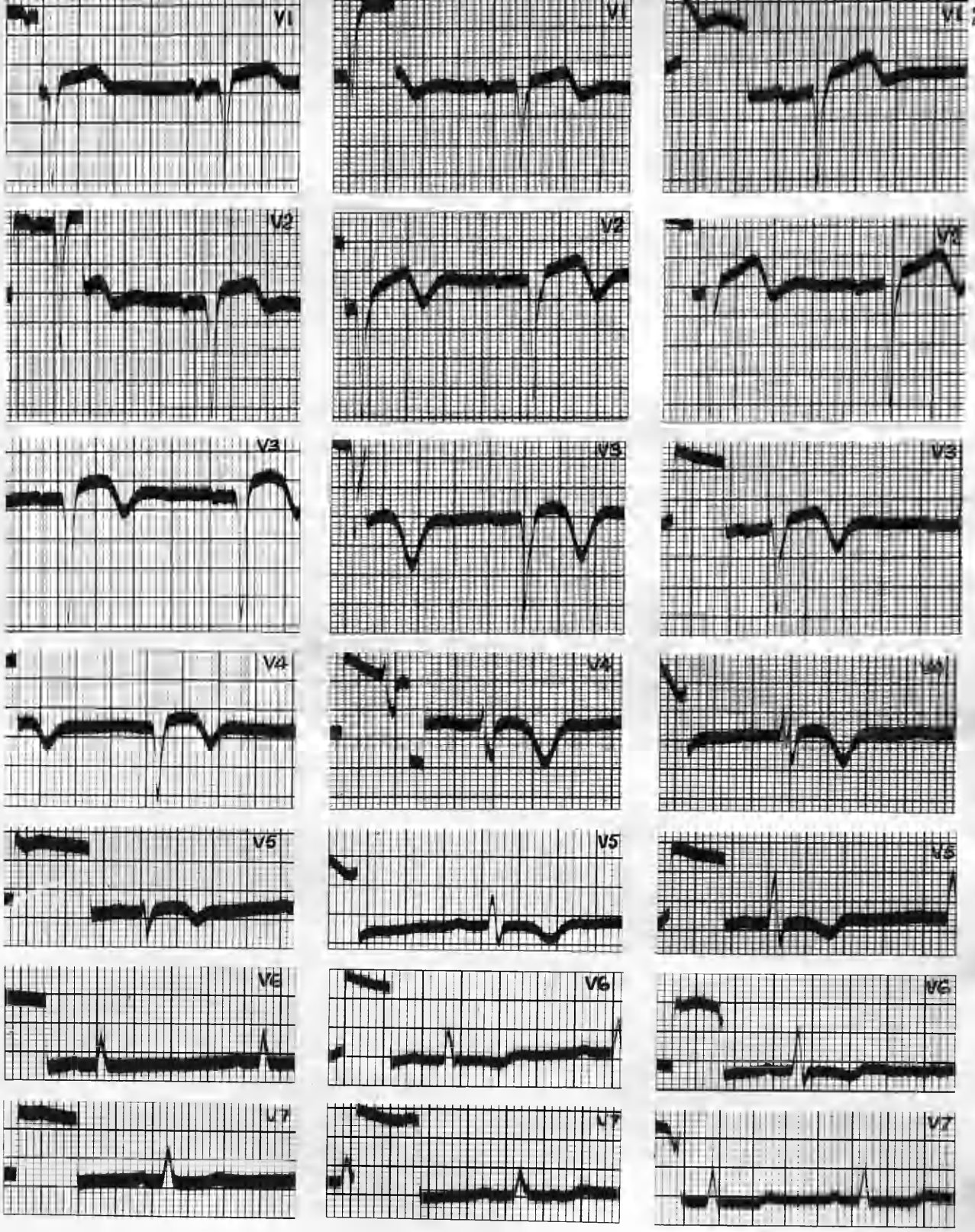


CASE 4, J.C., MALE, 53 YEARS.

6 DAYS AFTER ONSET.

7 WEEKS AFTER ONSET.

11 MONTHS AFTER ONSET.



CASE 4, J.C., MALE, 53 YEARS.

6 DAYS
AFTER ONSET.

7 WEEKS
AFTER ONSET.

11 MONTHS
AFTER ONSET.

Case 5, C.C., male, 36 years.

This man had a sudden attack of retrosternal radiating down the left arm one week before admission to hospital. His general condition was satisfactory. There was little evidence of shock and blood pressure on day of admission was 120/84, two days later it was 104/70 and thereafter it showed slight variations. Radiologically, the heart was normal in size and shape.

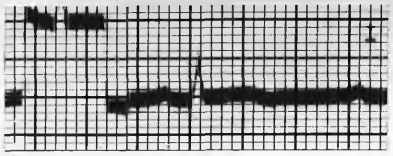
The electrocardiogram obtained on admission shows elevation of the ST segment in all three standard limb leads, especially in lead II. The T wave is small and is inverted in all leads but again chiefly in lead II. There is no Q wave in leads I and II but a small Q is present in lead III.

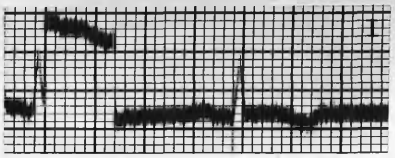
A deep QS deflection is presented by leads V1 to V3 inclusive; in lead V4 there is a sudden transition to a QRS deflection. Elevation of the ST junction, measuring two millimetres, is seen in leads V2 and V3 and one millimetre in lead V4. The ST segment retains its normal upward concavity in lead V2 but in leads V3 and V4 it shows straightening and upward convexity respectively, typical of the stage of organisation of infarction. Slight upward convexity persists in lead V5. Leads VR and VL show downward displacement of the ST segment and upright T wave typical of the intact left ventricular wall opposite an acute infarction.

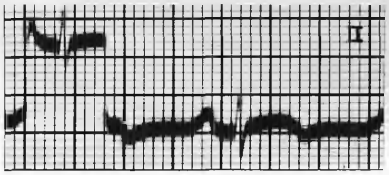
In addition the P waves are inverted. It is clear that both leads VR and VL face the back of the heart. Lead VF shows a very small ventricular deflection barely measuring five millimetres, but there is a very small Q wave which is as large as the following R wave. The ST segment is slightly elevated and the T inverted.

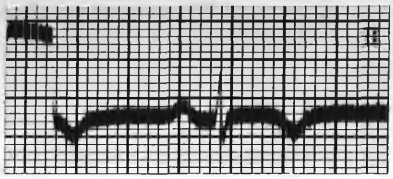
The praecordial leads indicate an anteroseptal infarction. Because of backward rotation of the apex the potentials from the lower edge of the infarction have been referred to the left leg and both right and left arms have recorded the reciprocal effects of the infarction. It is probable that the deep QS deflection of lead V1 indicates septal involvement and the signs at the V4 level are suggestive of subepicardial spread of infarction at the apex. The probable septal involvement may explain the splintered QRS of lead VL. The first small R wave may be due to positive potentials derived from activation of part of the septum from right to left and the large R wave which follows is the delayed intrinsic deflection of the lead.

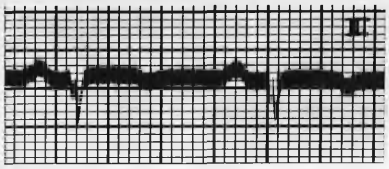
This case emphasises the importance of assessing cardiac position before estimating the size and position of an infarction and it shows how the classical signs in the standard limb leads are influenced by unusual cardiac positions.

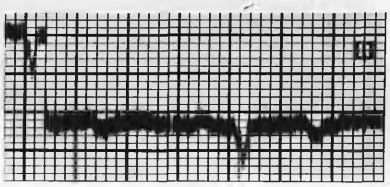


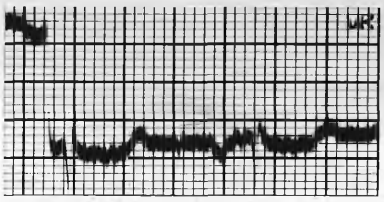


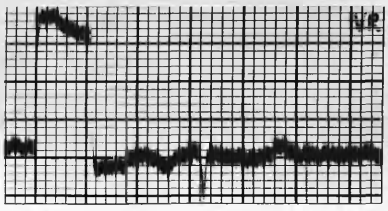


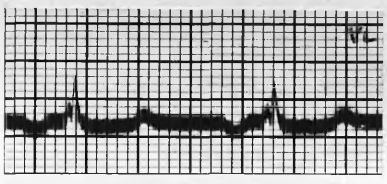


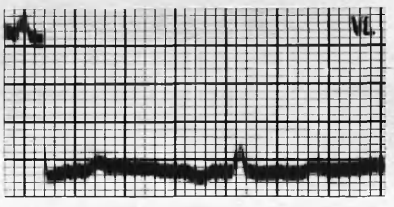


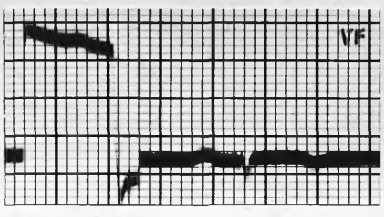


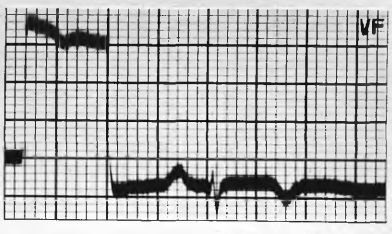








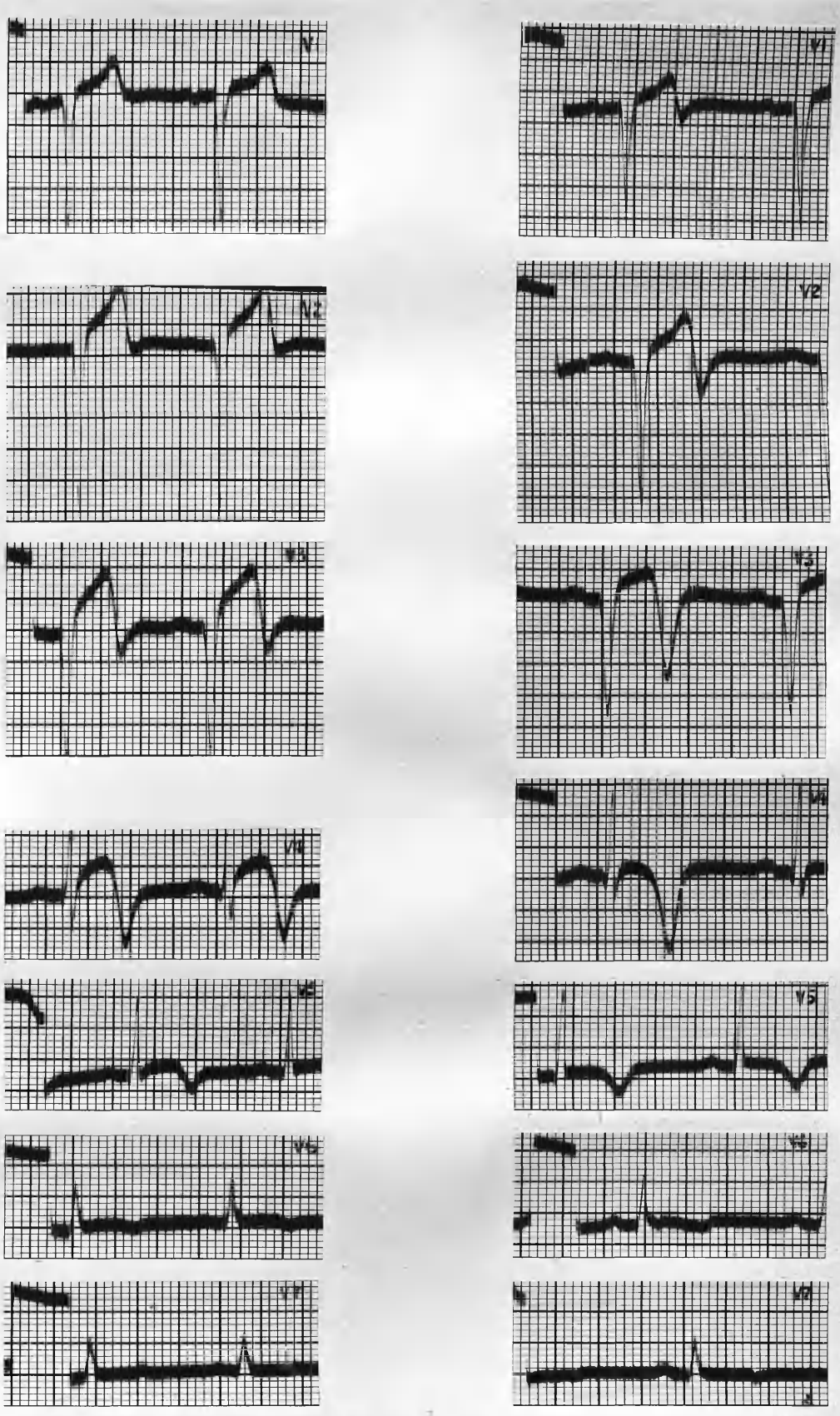




CASE 5, C.C. MALE, 36 YEARS.

1 WEEK
AFTER ONSET.

3½ WEEKS
AFTER ONSET.



CASE 5, C.C. MALE, 36 YEARS.

1 WEEK
AFTER ONSET.

3½ WEEKS
AFTER ONSET.

Case 6, J.L., male, 45 years.

This patient, a fitter by occupation, complained of pain over the praecordium spreading over the chest, which had lasted for two hours, three weeks prior to admission to hospital. The pain had been accompanied by sweating and vomiting. On the evening preceding admission it had returned, its situation being over the lower end of sternum with radiation over the chest. He recalled that two years previously, he had had severe pain in the praecordium, radiating over the chest and down both arms which lasted only a few minutes but which had recurred frequently for two or three weeks.

Patient showed no cyanosis or dyspnoea. There was slight elevation of temperature for several weeks. Pulse rate at first was 80 to 90, falling to 70 to 80 per minute: blood pressure on admission to hospital was 94/74 falling over the next ten days to 84/74 and thereafter slowly rising to 100/80. The cardiac sounds were soft; there were a few rales at the pulmonary bases.

The first 12-lead electrocardiogram was obtained three weeks after onset of illness. There is a small splintered R wave in standard limb lead I but no Q wave, which is not suggestive of infarction. The presence of a small S wave prevents any elevation of the ST-T junction. The slight plateauing of the ST segment and the inversion /

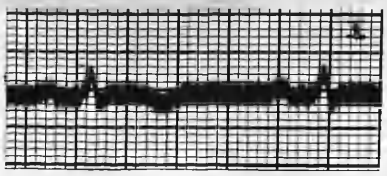
/ inversion of the T wave are however indicative either of infarction or of pericarditis. The reason for the paucity of signs is found in the unipolar limb leads. The deep single negative deflections of both leads VR and VL and the tall R wave of lead VF indicate that the heart is vertical and therefore lead VL is unfavourably placed to register central or marginal patterns from the infarct. There is however inversion of the T wave in lead VL which is faithfully reproduced in lead I. The plateauing of the ST segment is chiefly obtained from the reciprocal of the ST depression of lead VR which is however derived from the endocardial surface of the infarction.

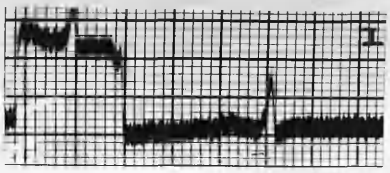
The praecordial leads show absence of the R wave in leads V1 and V2. In view of the absence of horizontal position it is unlikely that these leads are reflecting cavity potentials; because of their association with ST-T signs typical of infarction in other praecordial leads, the pattern in leads V1 and V2 is indicative of infarction of the anterior part of the septum and its terminus in the anteroseptal wall. The R wave reappears in leads V3 and V4; the latter shows a broadened and splintered S wave. Cove-plane T waves are seen in leads V3, V4 and V5 which is their usual position in anterior infarction. There is also slight upward convexity of the ST segment. The pattern in leads V3 and V4 would suggest infarction of the subepicardial layers only or /

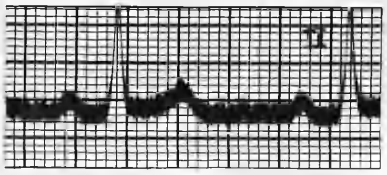
/ or pericarditis.

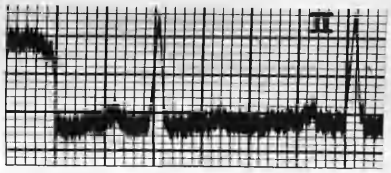
One year and three months later the limb leads show flat T waves in standard limb lead I and almost flat T waves in the other limb leads. The QRS complexes are typical of a vertical heart. The praecordial leads show persistence of the QS deflections in leads V1 and V2 and a similar QS is registered by lead V3 but their ST-T formations are now normal. Either there has been some shifting of the electrode at the V3 position or there has been spread of infarction at this point causing obliteration of the R wave, in the long interval between electrocardiograms. The persistent QS deflections in these leads no doubt represent the ultimate replacement of muscle by scar tissue at this stage. In lead V4 however there are signs of laminar recovery. There is still an early R wave but the previous notching of the broad S wave has developed into a prominent late R wave. Functional recovery is also reflected in the tall R waves of leads V5 and V6. There is still sharp inversion of the T waves of leads V4 and V5 but they are out of the region subtended by the left arm lead.

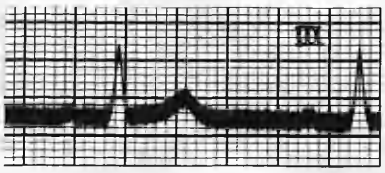
This case illustrates the scantiness of diagnostic signs of infarction in the standard limb leads largely due to vertical position of the heart. Signs of recovery and repair are evident, viz. left ventricular hypertrophy and laminar recovery at the left edge of the infarct.

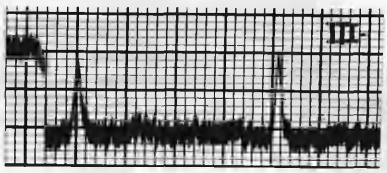


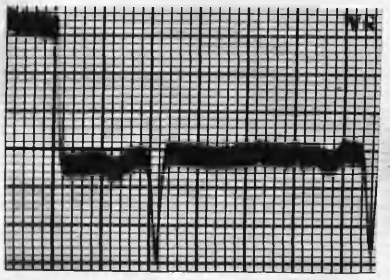


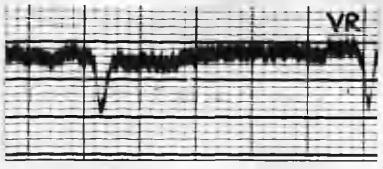


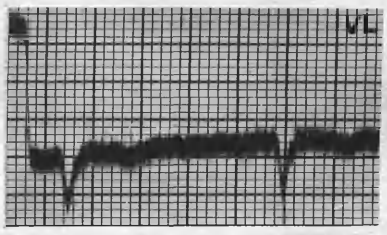


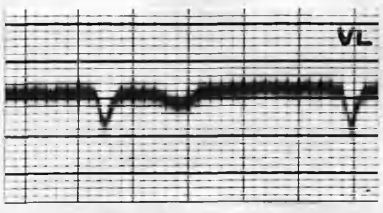


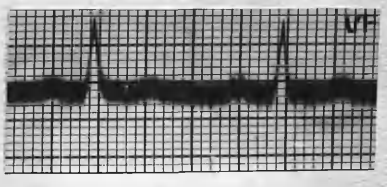


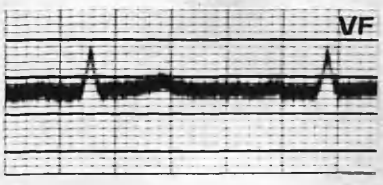








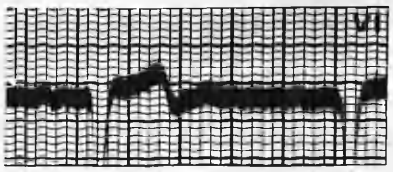


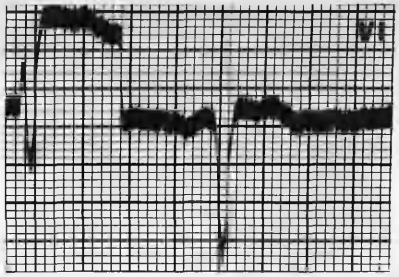


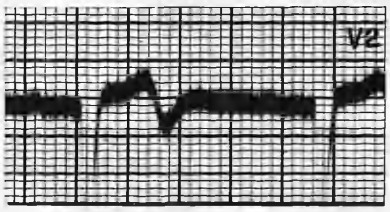
CASE 6, J.L., MALE, 45 YEARS.

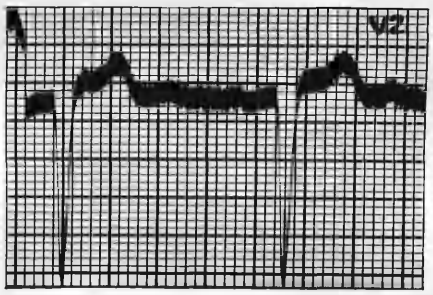
3 WEEKS
AFTER ONSET.

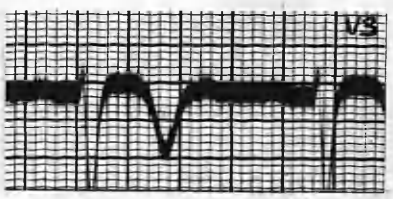
1 YEAR 3 MONTHS
AFTER ONSET.

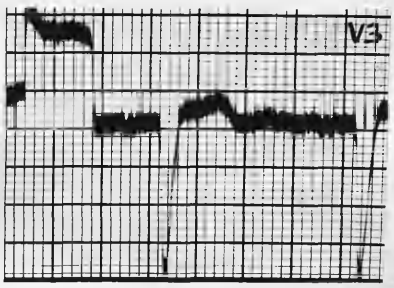


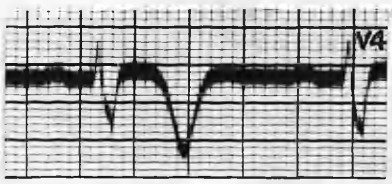


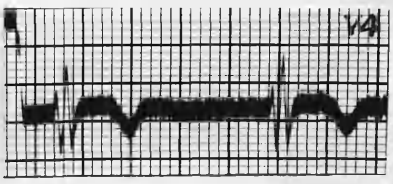


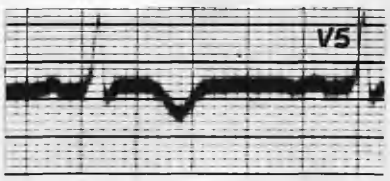


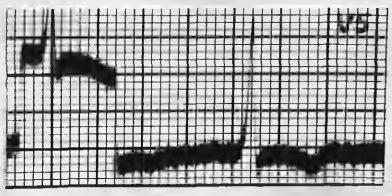


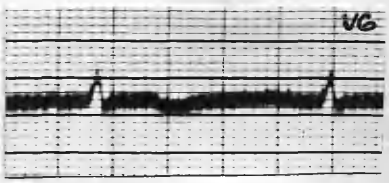


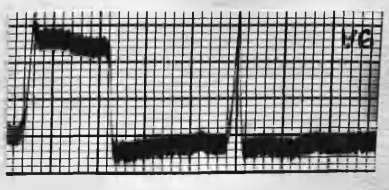












CASE 6, J.L., MALE, 45 YEARS.

3 WEEKS
AFTER ONSET.

1 YEAR 3 MONTHS
AFTER ONSET.

Case 7, D.M. male, 45 years.

This patient had his first attack of chest pain five weeks before he attended the out-patient department. The pain was of tearing character and lasted about an hour. A further attack occurred a week later radiating down the left arm and had persisted in milder form during the ensuing month. When examined at the out-patient department his blood pressure was 140 systolic, 90 diastolic. Apart from a deep sternal furrow the chest was of average shape. The apex beat was not palpable and the cardiac sounds were soft and toneless.

Standard limb lead I of the same date shows slight shouldering of the ST segment and a shallow inverted T wave, of a general contour undoubtedly suggestive of a recent anterior infarction. There is no Q wave but the R of the RS complex is relatively small. Lead I has clearly derived its potentials from lead VL and although in turn, its derivation from the praecordial leads is not clear it is probably related more to lead V5 than to lead V4 both of which however are over the zone of ischaemia. Leads V1, V2 and V3 show deep QS deflections which when viewed in association with the residual elevation of the ST segment in lead V3 and the cove-plane inverted T waves of leads V3 and V4, indicate a resolving anteroseptal infarction.

/ The patient was seen again six months later. He still had slight pain in the chest radiating down the left arm chiefly after heavier meals. Flatulence was troublesome.

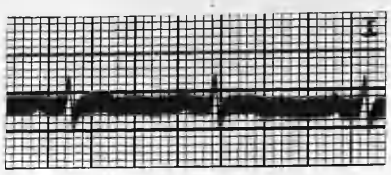
The standard limb leads, which, although abnormal, in so far as the QRS complex of leads II and III is broadened and notched, now show no frank sign of a previous infarction. Lead I no longer resembles lead VL which now shows signs derived from a marginal zone of infarction. A slight shift of cardiac axis may have occurred such that the marginal zone with its Q wave has been brought into the territory of lead VL in contradistinction to its previous reference limited to the zone of ischaemia.

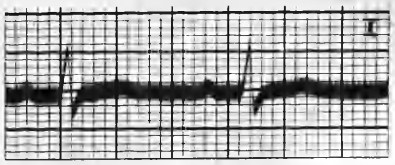
In view of the smallness of the deflections in lead VL, standard limb lead I has now derived its pattern largely from the reverse of lead VR. The praecordial leads show a very satisfactory regression of signs from the axillary leads forwards. Lead V4 as well as V5 are practically normal. The particular interest at this date centres on lead V3 where the R wave has returned, separating a Q from an S wave. This indicates some regeneration of active muscle elements in the left border of the infarction.

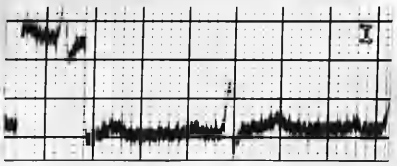
The last electrocardiogram was obtained more than two years from onset of illness by which time the patient had returned to work as a banker. Blood pressure was /

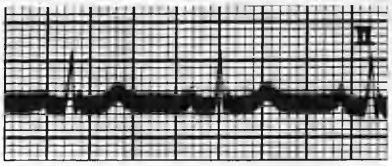
/was 116 systolic, 70 diastolic. There was very slight cyanosis but no dyspnoea. The chest was of average shape. The cardiac sounds were of average quality. Radiologically the heart was of normal size and pulsation was normal. Standard limb lead I and also lead VL are normal but the broadening and notching of the QRS deflection persists in standard limb leads II and III and in lead VF. A deep QS deflection persists as the only abnormality in leads V1 and V2. The boundary between scar tissue and active muscle is represented by the notched QS deflection of lead V3, the change in form from the previous electrocardiogram being no doubt due to slight change of position of the electrode and to the slight shift of cardiac axis. Leads V4 to V7 are normal.

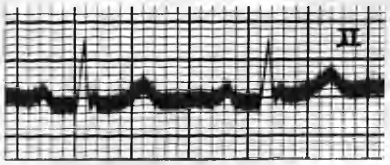
This heart was semivertical in position. The infarct had a broad ischaemic zone, the ST-T changes of which were readily referred to the left arm and therefore to lead I. On the other hand if the deflections in lead VL are small then the pattern of standard limb lead I may approximate much more closely to the reverse of its other component, viz. lead VR. In the final electrocardiogram the limitation of any abnormalities directly attributable to the infarction to leads V1, V2 and V3 explains the absence of such signs from any of the limb leads.

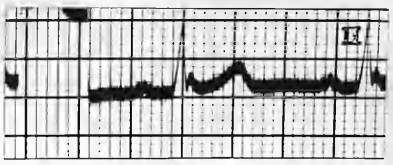


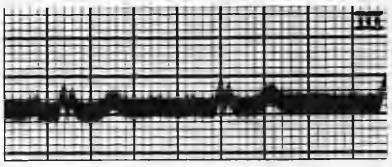


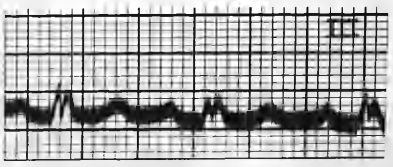


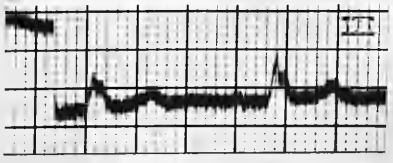


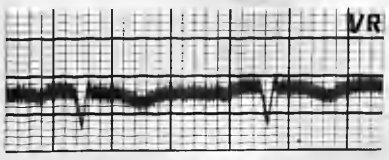


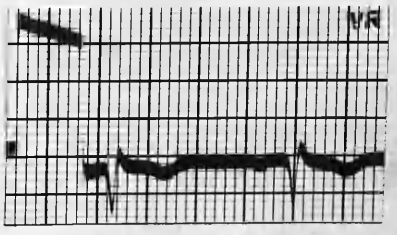


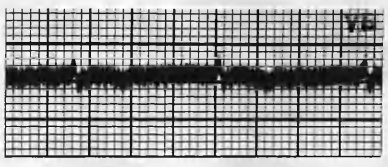


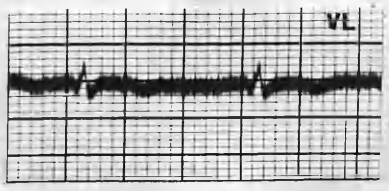


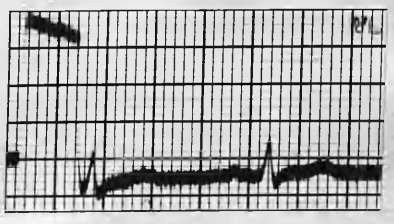


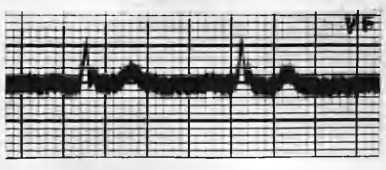


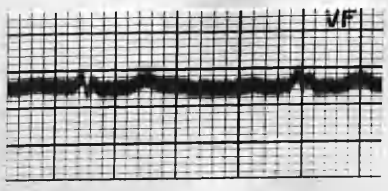


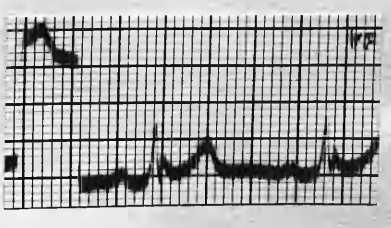










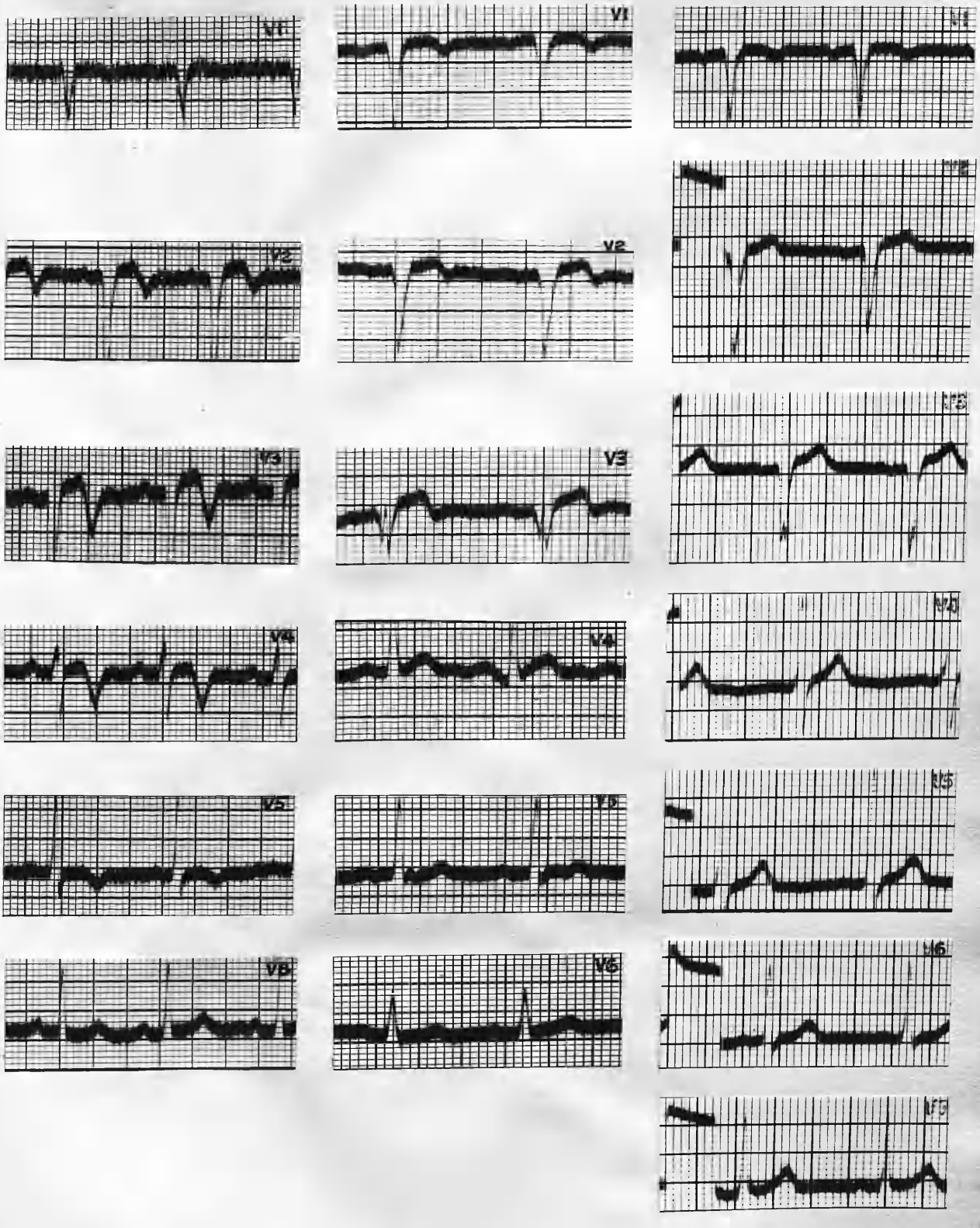


CASE 7, D.M. MALE, 45 YEARS.

5 WEEKS AFTER ONSET.

6 MONTHS AFTER ONSET.

2 YEARS 2 MONTHS AFTER ONSET.



CASE 7, D.M. MALE, 45 YEARS.

5 WEEKS AFTER ONSET.

6 MONTHS AFTER ONSET.

2 YEARS 2 MONTHS AFTER ONSET.

Case 8, W.W. male, 57 years.

This patient attended the out-patient department five weeks after onset of illness. He had had recurrent angina of effort for the previous month. The attack of pain which necessitated his confinement to bed had lasted two hours and had radiated widely over the chest and into both arms.

He was a powerfully built man. Blood pressure was 150 systolic, 80 diastolic. The cardiac sounds were of average intensity and pure.

Standard limb lead I shows slight upward bowing of the ST segment with shallow inverted T wave. The shape of this ST-T formation is very suggestive of anterior infarction; it has been derived from lead VL the deflections of which however are very small.

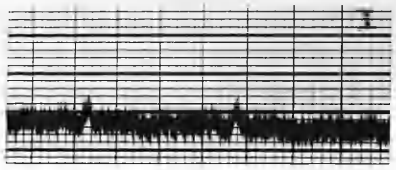
QS deflections are seen in V1, V2 and V3 and combined with the slightly elevated ST take-off and its upward convexity indicate an anteroseptal infarction with anterior septal spread. The RS pattern of lead V4 with shouldered ST segment and sharply inverted T wave indicate injury to the subepicardial zone of muscle only.

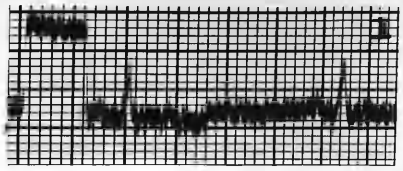
A year after onset the standard limb leads show shallow inverted T waves in lead I and diphasic T waves in lead II. Lead VL has a fixed marginal pattern with

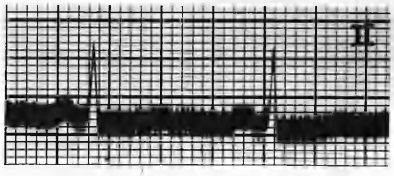
/ with a slurred prominent Q wave, a small R wave, plateaued ST segment and sharply inverted T wave. Lead VF shows a delayed intrinsic deflection indicative of a local conduction defect affecting the posterior wall.

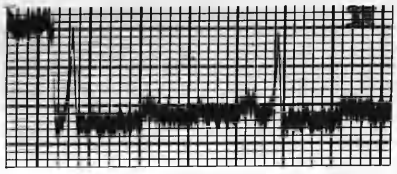
Leads V1 to V3 show persistent broad QS deflections and V2 to V5 inclusive still show deeply inverted T waves. The small upward notch in the short QS of lead V4 would suggest the presence of some active muscle tissue in this region. Leads V6 and V7 are in keeping with a minor degree of left ventricular dilatation.

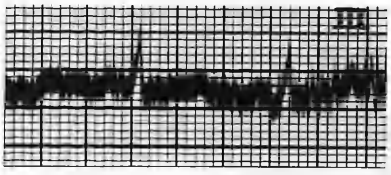
Clinically at this date the patient felt well. Blood pressure was 140/80 and the cardiac sounds were of average quality. Radiologically however there was "slight enlargement of the heart involving mainly the left ventricle"

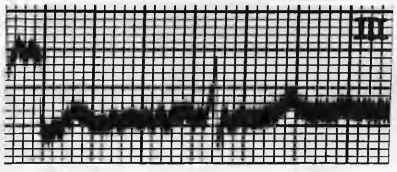


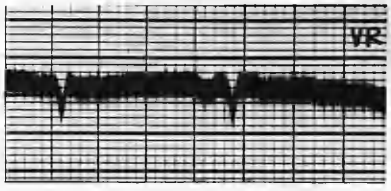


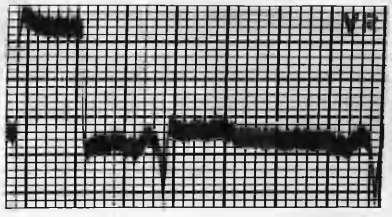


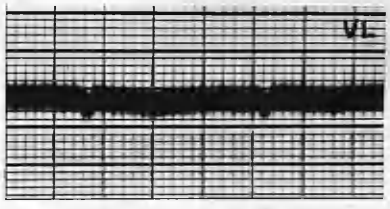


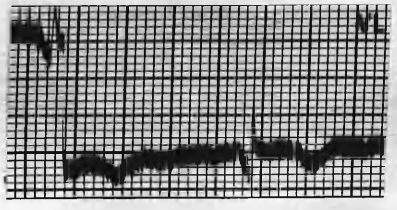


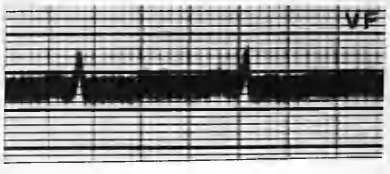


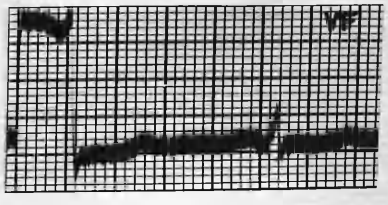








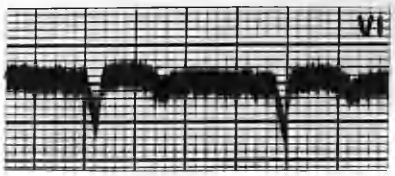


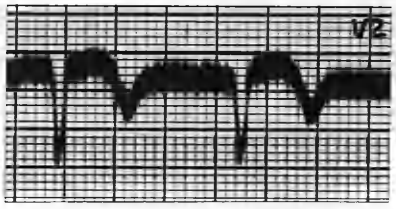


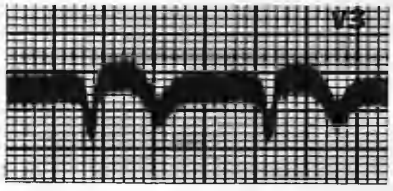
CASE 8, W.W., MALE, 57 YEARS.

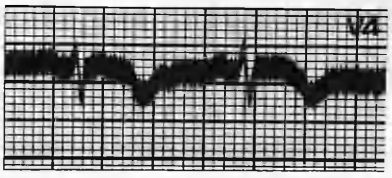
5 WEEKS
AFTER ONSET.

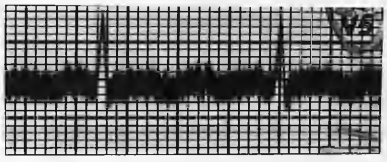
1 YEAR
AFTER ONSET.

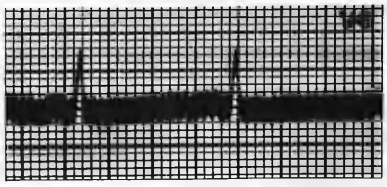


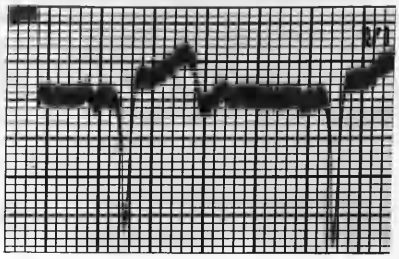


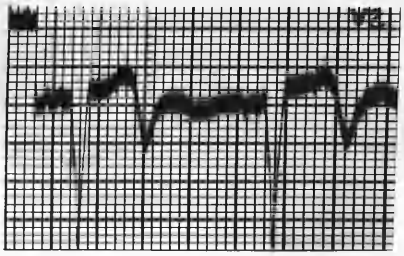


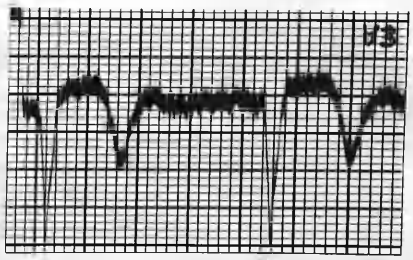


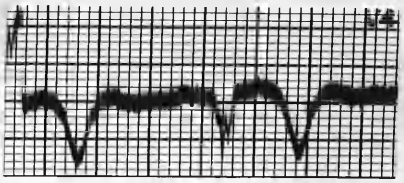


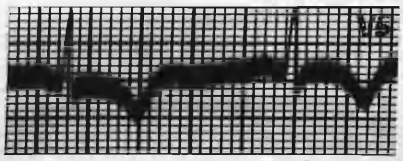


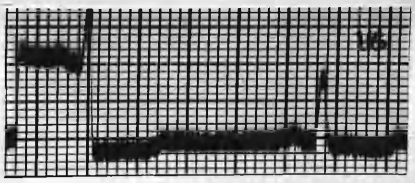


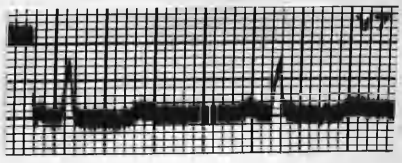












CASE 8, W.W., MALE, 57 YEARS.

5 WEEKS

AFTER ONSET.

1 YEAR

AFTER ONSET.

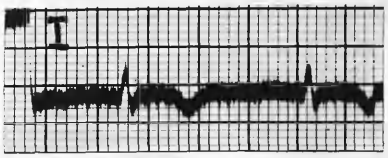
Case 9, F.H., male, 50 years.

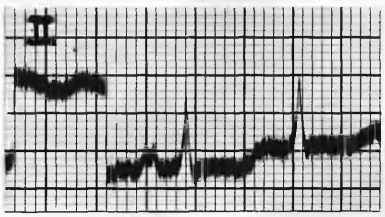
This patient had had recurrent anginoid pain dating from an acute attack which occurred nine months previous to his attendance at the out-patient department. The patient had been confined to bed at home for a month during the initial illness.

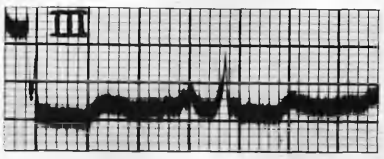
When examined at the out-patient department he showed normal blood pressure and no clinical signs of cardiac dilatation. The cardiac sounds were pure. Blood pressure was 150/100.

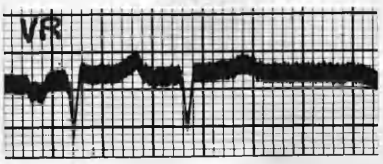
The electrocardiogram obtained at that time shows ST plateauing and inversion of the T wave very suggestive of previous anterior infarction. Lead VL shows frank signs of infarction, viz. a prominent Q wave, a very small R wave, ST elevation and inversion of the T wave but these signs have been mitigated by the reciprocal of lead VR which has cancelled the Q wave, increased the height of the R wave and decreased the ST elevation. The tall R wave of lead VF indicates a vertical position and the depression of the ST segment in this lead is typical of intact ventricular wall opposite an infarction. The praecordial leads show deep QS deflections in leads V1, V2 and V3 indicating previous infarction in the anterior part of the septum and adjacent anteroseptal wall. The /

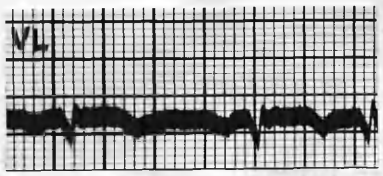
/ The ST-T deflections of these leads have returned to normal. In lead V4 there is a significant Q wave; in the first ventricular complex, which is of sinus origin, the Q wave is 25% of the succeeding R wave, in the second complex, which is a nodal extrasystole, it is almost 33% of the R wave. There is a deep S wave which is part of the transitional pattern between right and left ventricles. The ST segment is slightly convex upwards and there is a cove-plane T wave. Hence lead V4 clearly overlies the edge of the infarction. The ST-T formation suggests that organisation is still taking place. Hence the ST-T pattern in leads V5 and V6 probably indicates an ischaemic zone rather than left ventricular hypertrophy. Thus the praecordial leads demonstrate the residue from a previous septal and anteroseptal infarction, the extent and age of which cannot be assessed from the standard limb leads. At this late date, nine months after onset, the electrocardiographic signs are probably fixed.

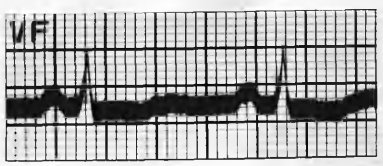


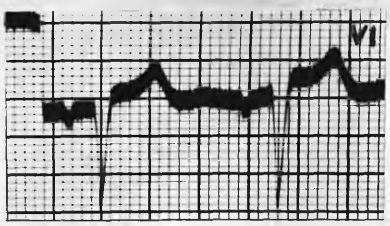


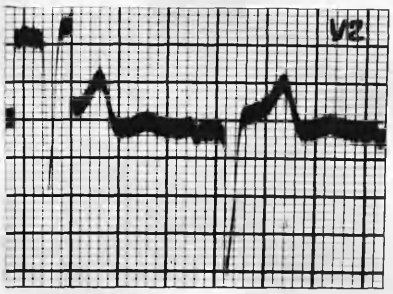


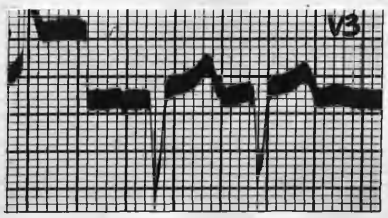


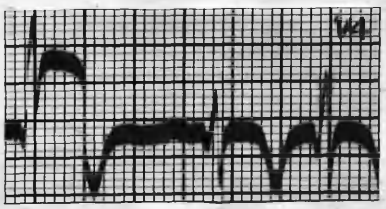


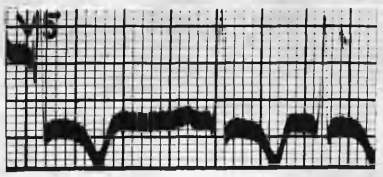


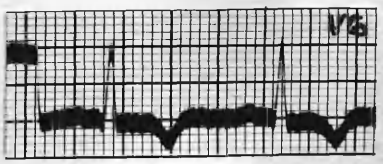












CASE 9, F.H., MALE, 50 YEARS.

9 MONTHS AFTER ONSET.

9 MONTHS AFTER ONSET.

Case 10, M.T., male, 43 years.

This patient had an attack of praecordial pain lasting eight hours six weeks before his attendance at the out-patient department. There had been no recurrence of pain. Blood pressure was 100/70 and the cardiac sounds were soft. An electrocardiogram shows a very small Q wave in lead I and a sharply inverted T wave. The ST-T contour is suggestive of a previous anterior infarction. Deep cove-plane T waves are seen in leads V3 and V4 and lesser degrees of inversion in leads V2 and the axillary leads. There is a very small R wave in lead I indicating intact septal function but there is no R wave in leads V2 and V3. The splintered QRS formation in lead V4 suggests that the infarction is laminar and not transmural at this situation.

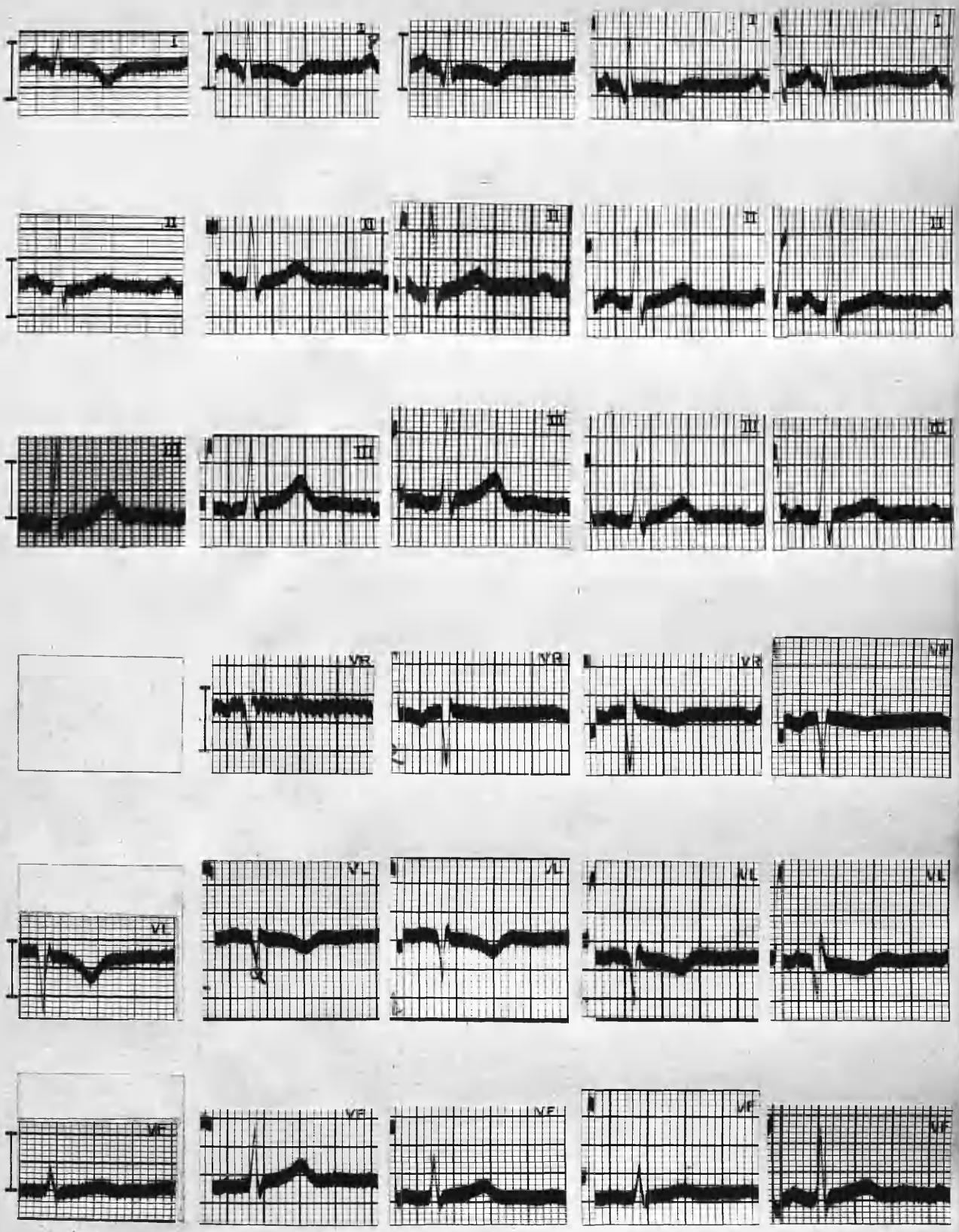
The pattern of lead V1 which shows a prominent R wave would suggest derivation from the anterior cardiac wall in the region of the conus pulmonalis, the late activation of which would inscribe the R wave. Thus there is probably forward rotation of the apex beat of this vertically placed heart. The next set of electrocardiograms shows satisfactory regression of the T waves in leads V1, V2 and V3 and also in V6 but in leads V4 and V5 there is still sharp inversion. Deep QS deflections typical of anteroseptal infarction persist in leads V2 and V3 and appear for the

first time in lead V4 where previously the infarction was laminar. In view of the ease of placing the electrode on the apex-beat, which was palpable in this case, the change in lead V4 is probably not entirely due to shift of position of the electrode.

Three months after onset there is still little change in the standard limb leads. The only new features in the praecordial leads are the development of a conspicuous notch towards the end of the QS deflection in lead V4 and the slight delay in attaining the peak of the R wave in leads V5 and V6. The notch in lead V4 becomes a positive late R wave two months later and by one year and eight months after onset it has become a conspicuous late upward deflection signifying recovery of function in the subepicardial layers at this level. Similarly, lead VL registers a new late R wave since by forward rotation of the apex lead VL derives its potentials from the anterior cardiac wall. Leads V5, V6 and V7 and also VF now show the signs typical of left ventricular hypertrophy or dilatation, viz. increased height of the R wave and in the axillary leads sagging of the ST segment and shallow diphasic T waves; in addition there is increased depth of the QS deflection in leads over the right side of the praecordium. Although the ST-T deflections of leads V2 and V3 have returned to normal there is still a

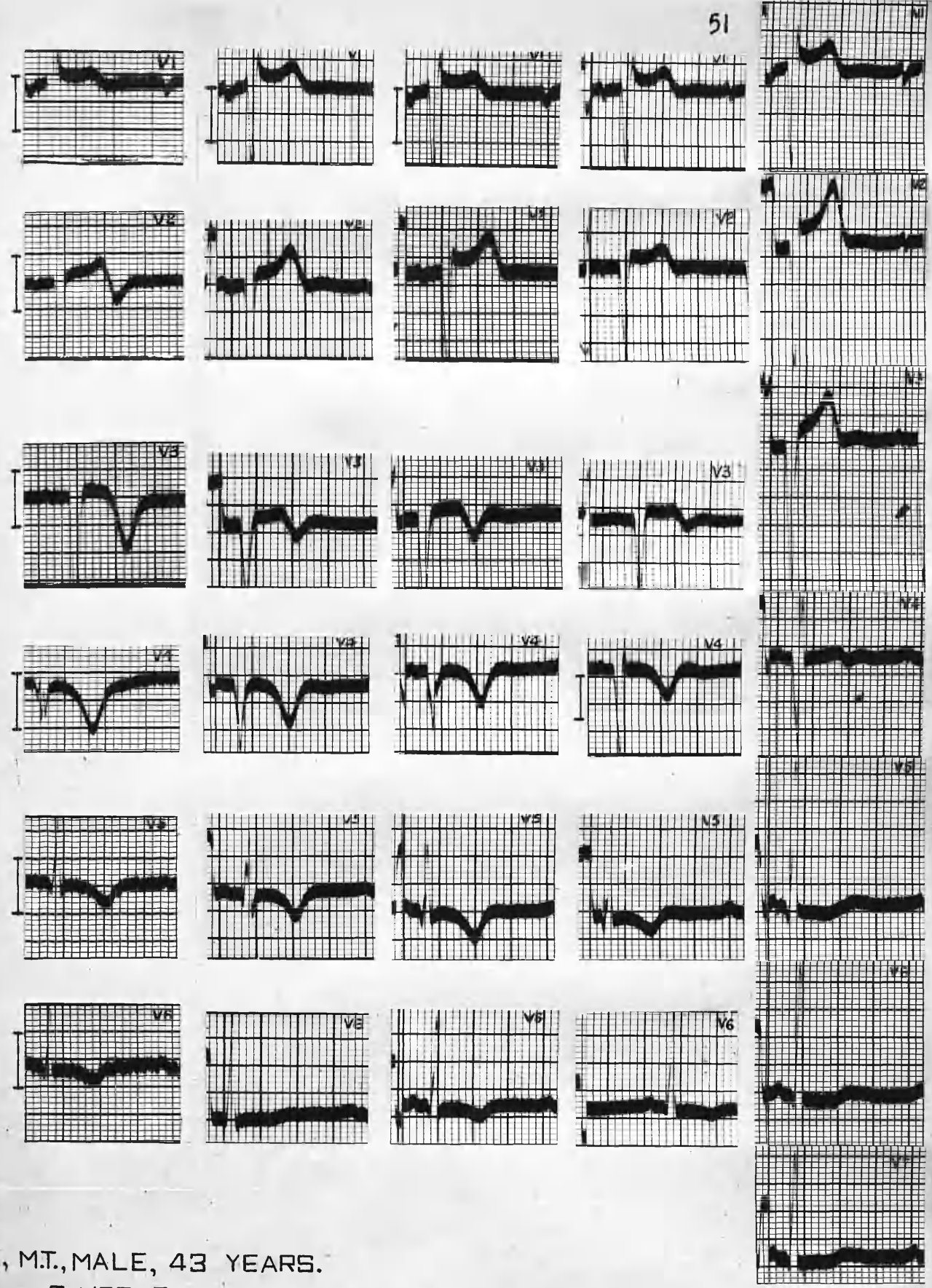
deep QS deflection in both leads. Thus the last electrocardiogram represents a combination of various features - persistent signs of infarction viz. the absence of an early R wave in leads V2 and 3 and V4 - no doubt an indication now of scar tissue - signs of compensatory hypertrophy or dilatation of the left ventricle and signs of repair revealed by the new late R wave in lead V4.

It is to be noted that the R wave of leads V5 and V6 is a large upright deflection as early as six weeks after onset which would suggest that the lateral wall of the left ventricle was practically intact and was therefore a likely region to initiate compensatory changes.



No 10, M.T., MALE, 43 YEARS.

6 WEEKS AFTER ONSET.	10 WEEKS AFTER ONSET.	3 MONTHS AFTER ONSET.	5 MONTHS AFTER ONSET.	20 MONTHS AFTER ONSET.



No 10, M.T., MALE, 43 YEARS.

6 WEEKS AFTER ONSET.	10 WEEKS AFTER ONSET.	3 MONTHS AFTER ONSET.	5 MONTHS AFTER ONSET.	20 MONTHS AFTER ONSET.

Case 11, S.K. male, 44 years.

This patient, a railwayman, had been confined to bed at home for several weeks because of recurring attacks of retosternal constriction and numbness of the left arm which had occurred two or three times daily for a few weeks and were accompanied by weakness and sweating.

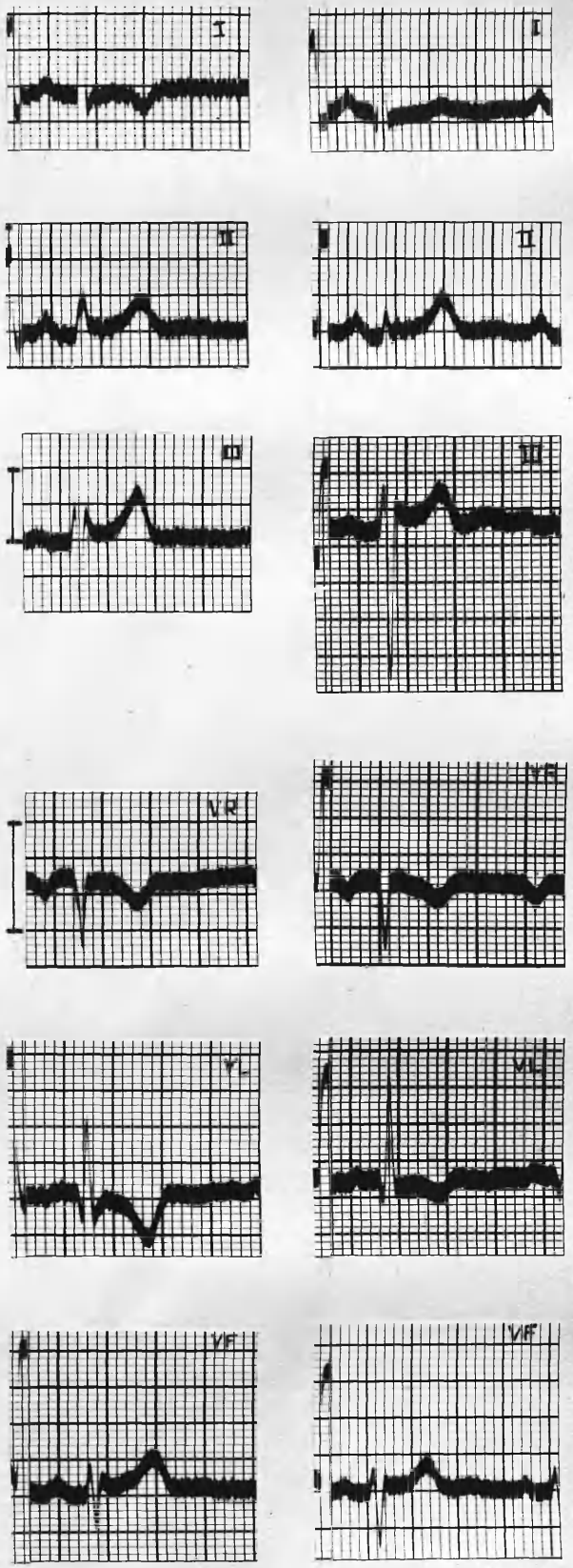
When eventually seen at the out-patient department, the cardiac sounds were of average quality. Blood pressure was 150/90. There was no clinical cardiac enlargement. The chest was of broad deep shape. Radiologically the heart showed a hypertensive configuration with some enlargement of the left ventricle. An electrocardiogram taken at this time shows left axial deviation in the standard limb leads. There is upward convexity of the ST segment and inversion of the T wave suggestive of recent anterior infarction in lead I. This contour is derived from lead VL where there is however a Q wave which is twenty-five per cent of the succeeding prominent R wave. Thus subendocardial infarction of the lateral wall of the left ventricle is suggested. The Q wave has not been transmitted to standard limb lead I because of the simultaneous negative deflection in lead VR. In the praecordial leads the first phase of the ventricular complex is abnormal in only two leads, viz. leads V1 and V2. A small Q wave followed by an equally small R wave

/ R wave is seen clearly in lead V2 and less conspicuously in lead V1. A Q wave in these leads is due to initial activation of the septum from right to left and not from left to right which is the normal early direction of septal potentials. The small R probably represents potentials arising in the free wall of the right ventricle. Leads further to the left show a transitional RS pattern or a left ventricular pattern but there is no Q wave in leads V3 to V6 inclusive. However the shape of the ST segment and the cove-plane T waves are very typical of an anteroseptal infarction; the deepest inversion is seen in lead V4 where it reaches sixteen millimetres. It is probable therefore that the region of the free wall of the left ventricle actually infarcted was at a higher level than the sites of these praecordial leads, especially in view of the fact that lead VL records a significant Q wave. The alternative explanation for the Q wave is that it is the result of the normal activation of the unaffected posterior part of the septum. Thus septal infarction can be diagnosed with some certainty and there is a high probability that the anteroseptal wall is also involved.

Patient was again examined more than a year later. He stated he felt well although he still had upper sternal pain radiating into the neck on walking. Exertional dyspnoea was also present. The build was stocky and the /

/ the chest broad and deep, as before. The heart sounds were soft. Blood pressure was 156/100. The left shoulder joint was painful on movement. An electrocardiogram now shows severe left axial deviation. There is now a small Q wave in standard limb lead I derived from lead VL where it is now early enough to evade cancellation by the negative deflection of lead VR. It is clearly a normal septal Q in lead VL and therefore it was probably of similar nature in the first tracing. The residual signs of the septal infarction can be detected in leads V1 and V2. The T waves of the praecordial leads have regressed almost to normal.

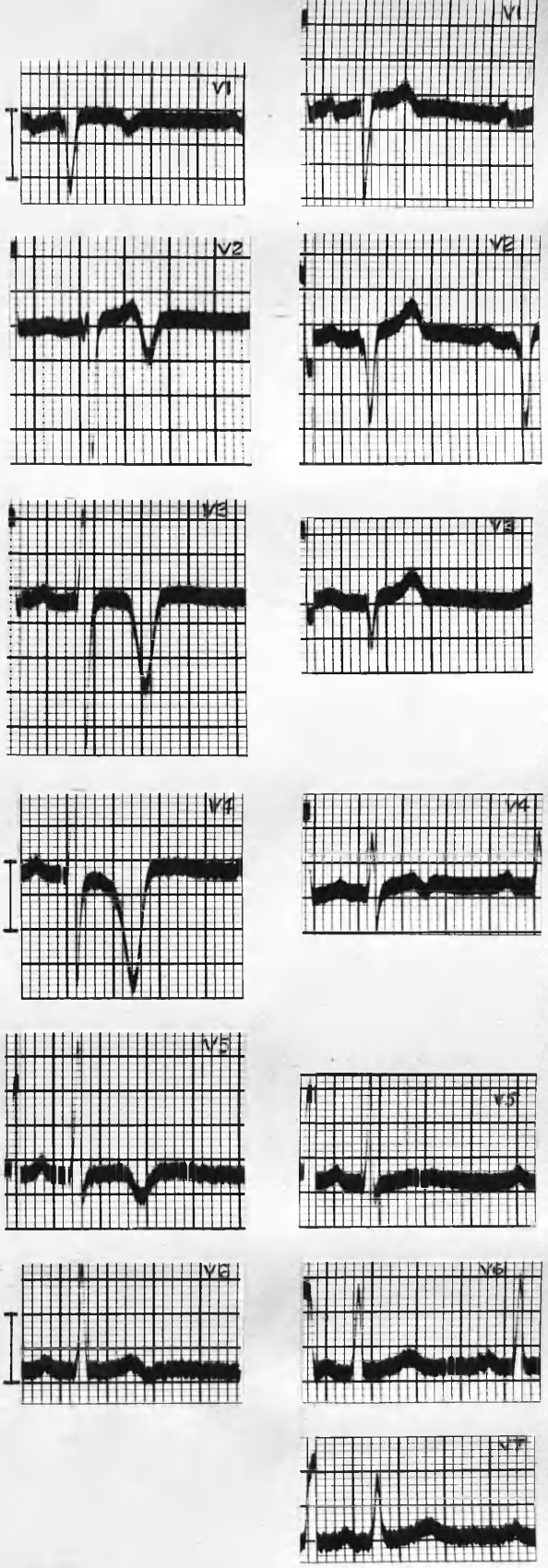
This case is an example of a septal or more probably septal and anteroseptal infarction occurring in a horizontal heart due to left ventricular hypertrophy.



No 11, S.K., MALE, 44 YEARS.

2½ MONTHS AFTER ONSET.

16 MONTHS AFTER ONSET.



No 11, S.K., MALE, 44 YEARS.

2½ MONTHS AFTER ONSET.

16 MONTHS AFTER ONSET.

Case 12, S.W., male, 51 years.

This patient, a steel-worker, first attended the out-patient department six weeks after an attack of anginoid pain lasting several hours. General condition was good. The cardiac sounds were soft and B.P. was 116/70. Radiologically the size of the heart was normal although the left border was prominent.

Electrocardiograms taken six weeks after onset show shallow inversion of the T waves in lead I as the sole evidence of possible anterior infarction. The praecordial leads however show deep QS delections in leads V1 to V3 inclusive, combined with deep cove-plane T waves indicating anteroseptal infarction. In lead V3 and T inversion is deepest, measuring thirteen millimetres.

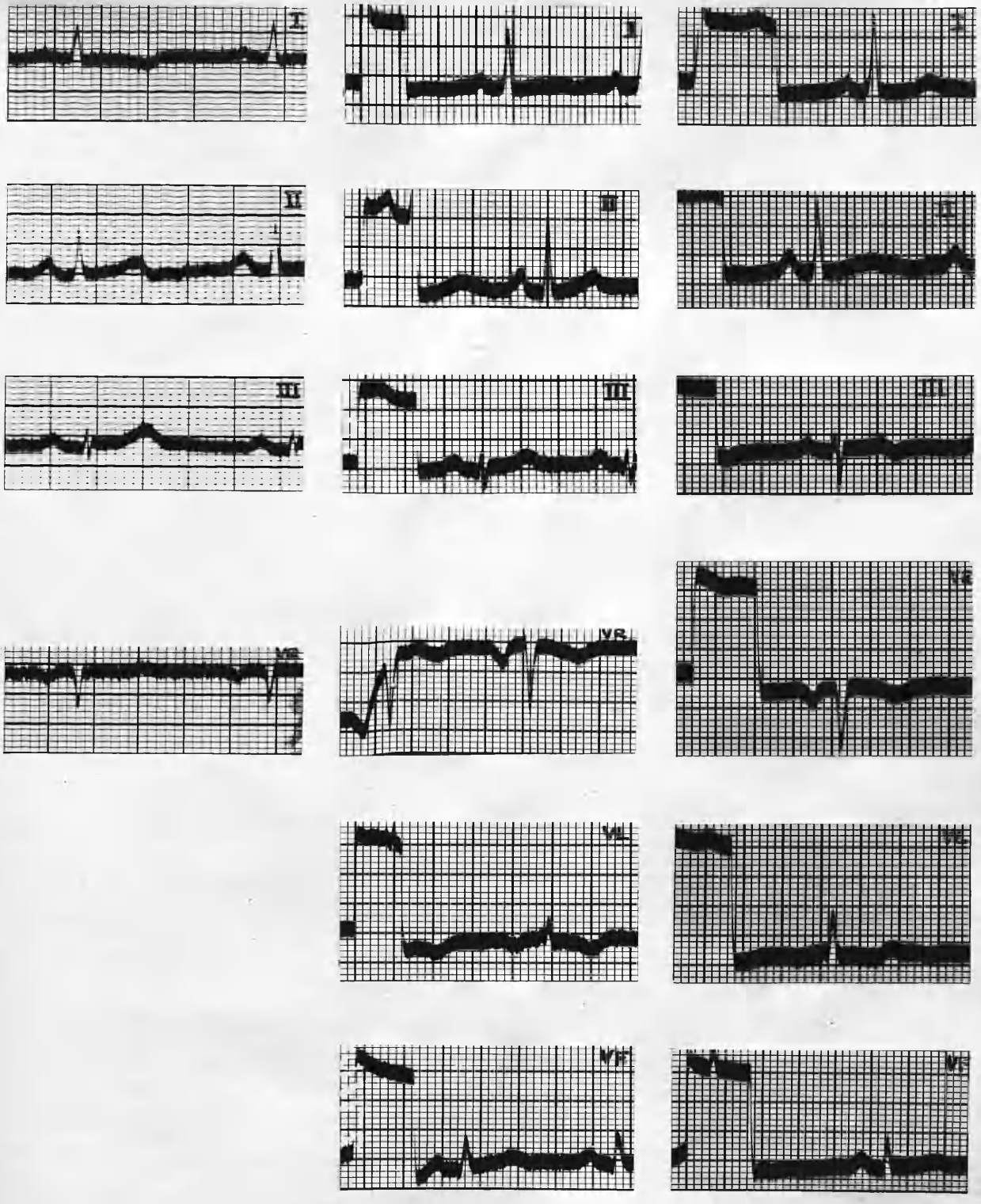
Recovery was uneventful. When seen again eight months after onset the cardiac sounds were soft; B.P. was 120/60. Eight months after onset the ST-T segment of standard limb lead I is practically flat. While allowance must be made for shift of electrode the pattern of lead V4 would now suggest further subendocardial infarction in the region of the apex. There is a Q wave which is very probably significant of subendocardial involvement because it measures at least twenty-five per cent of the succeeding R wave and because there is no Q wave in leads further to

the left. The upward ST bowing is highly suggestive of a recent lesion. In the other praecordial leads, inversion of the T wave has almost disappeared but QS deflections persist in leads V1 to V3 inclusive. The unipolar limb leads indicate that the heart is in the intermediate or semivertical position so that the absence of an R wave in lead V1 is probably due to involvement of the anterior part of the septum.

The final tracing taken one year and eight months after onset shows frank left axial deviation in the standard limb leads. The unipolar limb leads indicate that the heart is now in the intermediate position. The end of the T wave of leads V1, V2 and V3 is inverted but the prominent U wave of these leads gives a fallacious impression of this inversion. Otherwise there is no ST-T abnormality in the praecordial leads. Deep QS deflections persist however in leads V1, V2 and V3. The Q wave previously seen in lead V4 has now disappeared. The apical and axillary leads show increased height of their R waves which is either due to the assumption of the intermediate position or to early left ventricular hypertrophy. There is no sign of regeneration at the left edge of the infarct where it is most likely to occur.

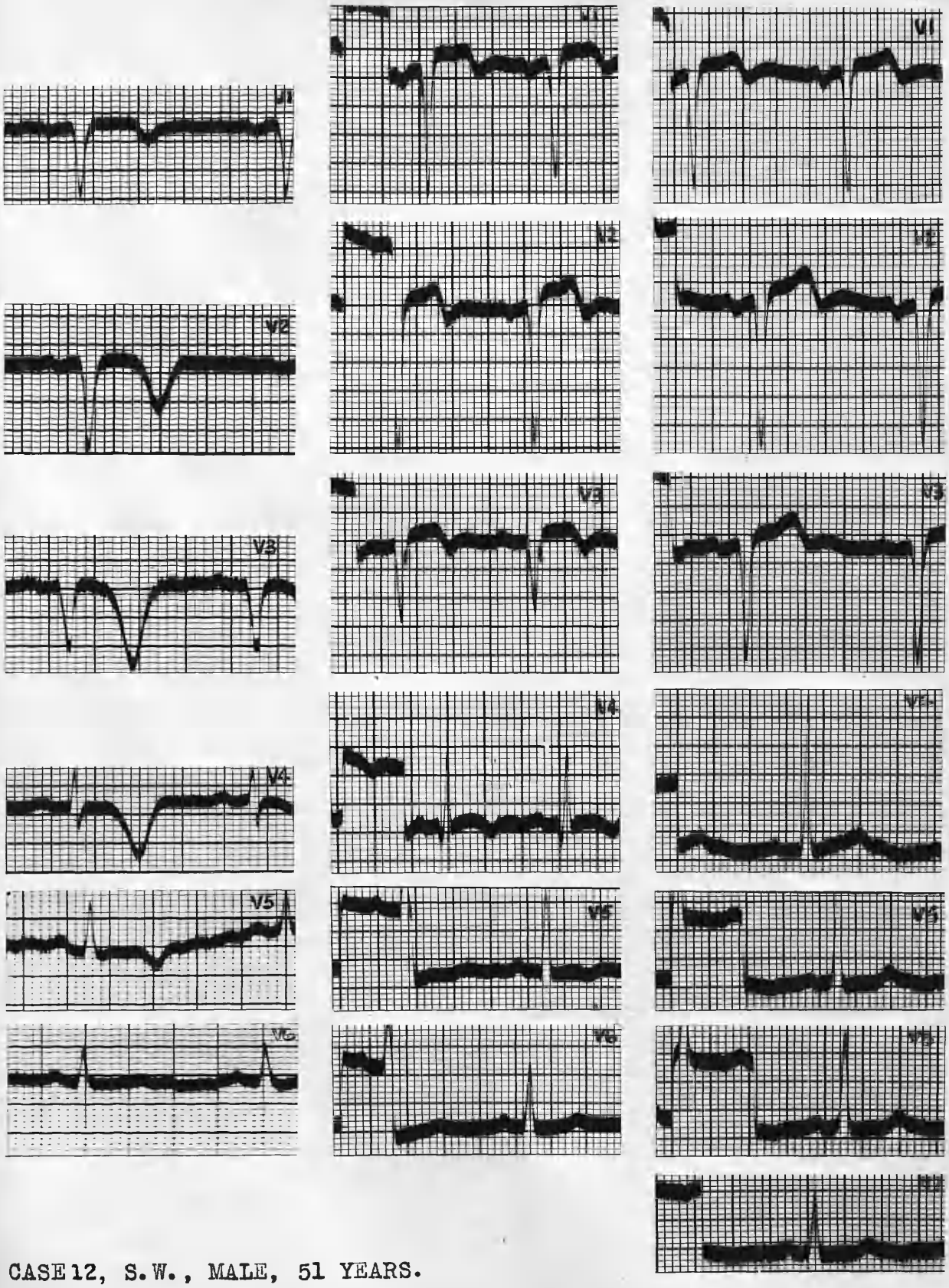
At this date (one year and eight months after onset) the patient felt and looked well. B.P. was 130/80. The

The chest was of average form and the apex beat was palpable in the 5th left interspace in mid-clavicular line. The cardiac sounds were of average intensity and pure. Radiologically there was obvious increase in size of the cardiac shadow, especially to the left (see facsimiles). These findings are consistent with the electrocardiograms.



CASE 12, S.W., MALE, 51 YEARS.

6 WEEKS AFTER ONSET.	8 MONTHS AFTER ONSET.	1 YEAR 8 MONTHS AFTER ONSET.

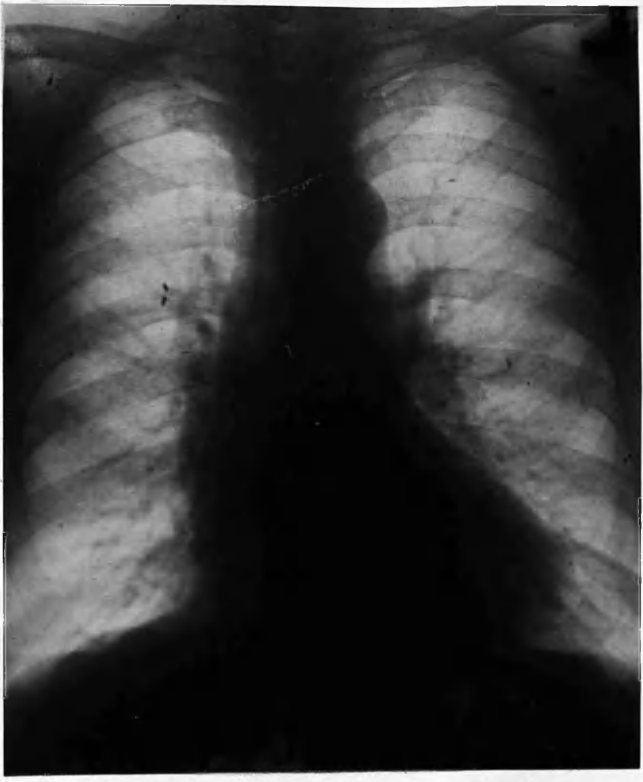


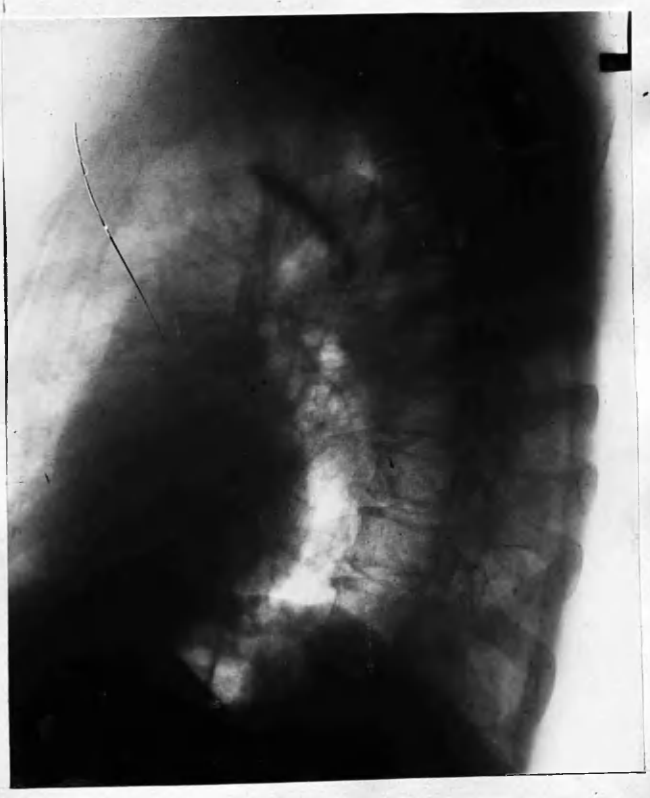
CASE 12, S.W., MALE, 51 YEARS.

6 WEEKS AFTER ONSET.

8 MONTHS AFTER ONSET.

1 YEAR 8 MONTHS AFTER ONSET.





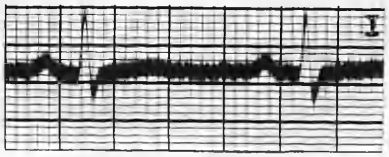
Case 13, W.L. male, 51 years.

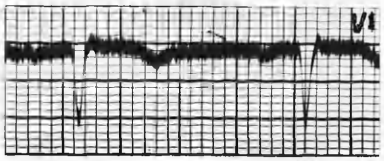
This patient attended the out-patient department five and a half weeks after an attack of chest pain which lasted twelve hours. There was no clinical enlargement of the heart; the cardiac sounds were soft and pure; blood pressure was 120/70.

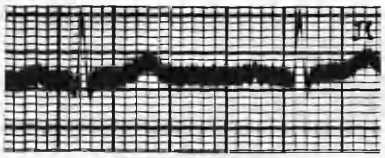
An electrocardiogram shows a low upright T wave in standard limb lead I; there is a prominent Q wave in lead III which is not necessarily abnormal in this lead. The unipolar limb leads show slight upward convexity of the ST segment and very shallow inversion of the T wave in lead VL but these have largely been cancelled out in lead I by the reverse direction of the ST-T contour derived from lead VR. Lead VF shows a small ventricular complex just reaching five millimetres and consisting of a small Q wave which is however half of the size of the succeeding R wave. It is probable that this QR formation is derived from the marginal zone of infarction by slight backward rotation of the cardiac apex. The Q wave of lead VF added to the reciprocal of the R wave of lead VL accounts for the prominent Q wave of standard limb lead III.

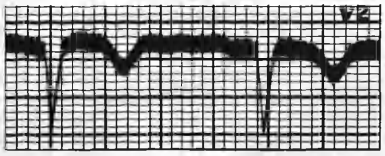
The praecordial leads show deep QS deflections in leads V1, V2 and V3. A notch early in the downstroke of the QS in lead V3 is clearly the homologue of the very

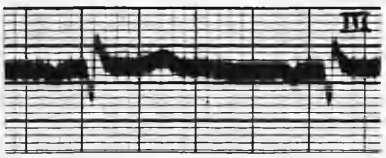
/ very small R wave which appears in lead V4, separating a small Q from a deep S wave; this represents a marginal pattern. Cove-plane T waves are prominent in leads V2, V3 and V4, deepest in V3. The praecordial leads are thus typical of an anteroseptal infarction in the stage of organisation. The question as to whether the deep QS deflection in lead V1 represents septal involvement or a normal variant in a horizontal heart must rest on the assessment of cardiac position. The pattern of lead VF would suggest an origin from the left rather than the right ventricular surface and hence that the cardiac position is intermediate to vertical with, probably, some backward rotation of the apex, such that a marginal pattern was transmitted to lead VF. Hence the QS deflection of lead V1 probably represents septal involvement.

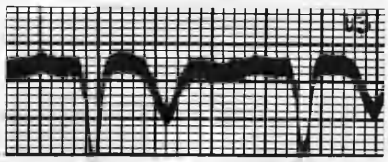


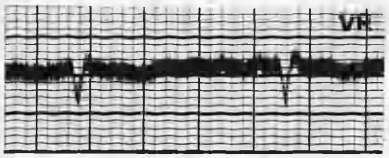


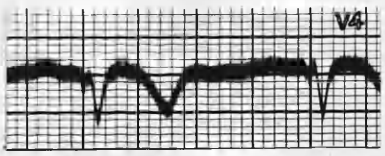


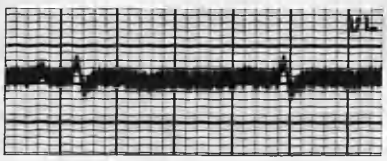


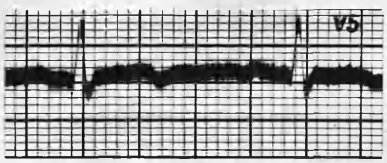


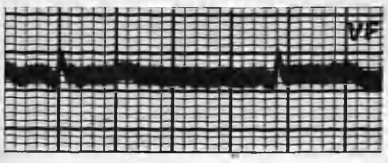


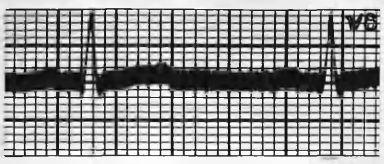












CASE 13, W.L.,MALE, 51 YEARS.

5½ WEEKS
AFTER ONSET.

5½ WEEKS
AFTER ONSET.

Case 14 H.W., male, 55 years.

This patient, a coal-filler by trade, was seen at the out-patient department four weeks after a severe attack of substernal pain which had confined him to bed at home for three weeks. Blood pressure was 128/80. The cardiac sounds were of average intensity and pure.

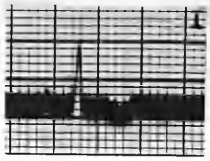
An electrocardiogram shows inversion of the T wave in standard limb lead I as its sole abnormality. The unipolar limb leads are slightly more informative in so far as there is a minute Q wave, a small R wave, slight shouldering of the ST segment and inversion of the T wave in lead VL, which constitutes an incomplete marginal pattern of infarction. The unipolar praecordial leads are indicative of anteroseptal infarction; there is a deep QS deflection in leads V2 and V3. The interpretration of the deep QS deflection in lead V1 depends on the electrical position of the heart. It occurs as a normal variant in the horizontal electrical position but it is improbable that such was the position of the heart in this case. Although the assessment of cardiac position is always hazardous in the presence of infarction, the chief ventricular deflection in both leads VL and VF is an R wave which would suggest the intermediate position in this case. Hence the deep QS deflection of lead V1 is probably pathological and

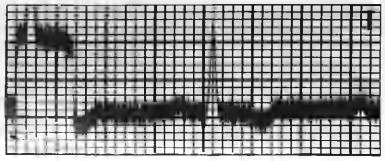
indicates involvement of the left side of the anterior terminus of the septum. The apical lead V4 has a tall R wave preceded by a minute Q wave. Although the Q/R ratio is well within normal limits, the Q wave is not repeated in leads further to the felt. Besides the Q wave of lead V4 is accompanied by shouldering of the ST-T segment and inversion of the T wave, which would suggest a derivation from the periphery of the infarct.

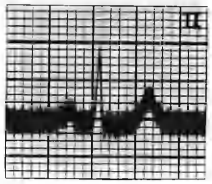
Ten months later, the patient was examined again. He still complained of pain in the front of the left chest accompanied by dyspnoea on walking, especially after a meal. The pain would radiate down both arms. He was slightly cyanosed. Blood pressure was 104/60. The apex-beat was faintly palpable in normal situation and the cardiac sounds were pure.

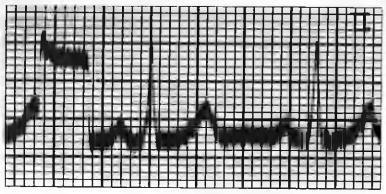
The only change in the standard limb leads is the development of a small Q wave in standard limb lead I, derived from the prominent Q wave which has appeared in lead VL. Thus lead I remains suggestive of a previous anterior infarction. The praecordial leads show signs of partial healing. A tiny R wave has returned to lead V1 but leads V2 and V3 show the stigma of previous infarction in persistence of a deep QS deflection. However a new late R wave has appeared in lead V4 homologous to the heightened R wave of lead V4

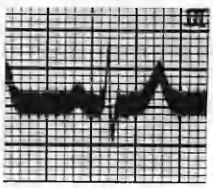
and indicative of laminar recovery in the subepicardial layers. The Q wave of lead V4 is now clearly pathological - it is thick and splintered and lasts 0.04 sec. It would indicate persistent involvement of the subendocardial layers.

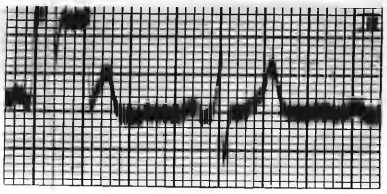


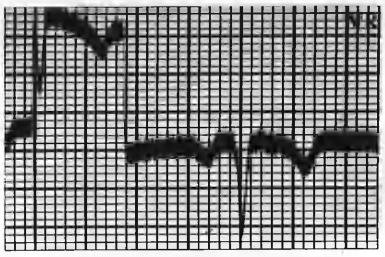


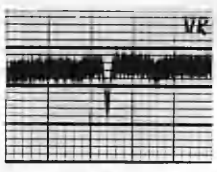


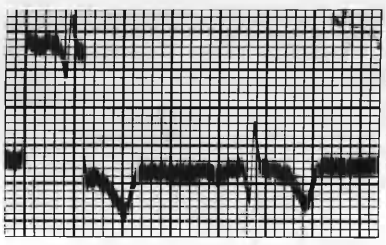


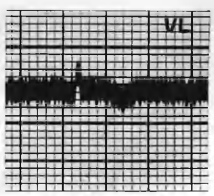


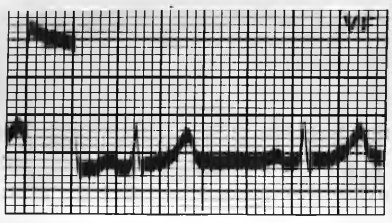


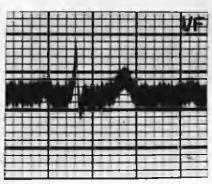








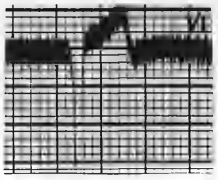


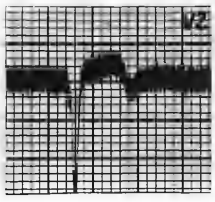


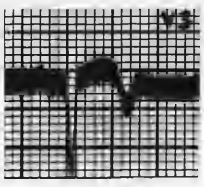
CASE 14, H.W. MALE, 55 YEARS.

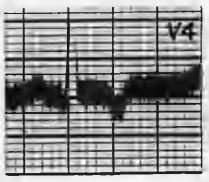
4 WEEKS
AFTER ONSET.

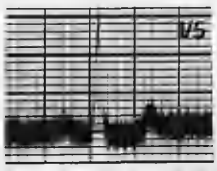
11 MONTHS
AFTER ONSET.

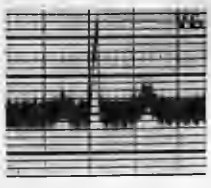








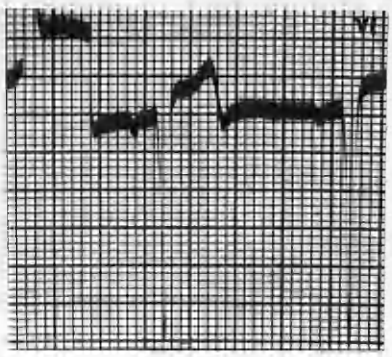


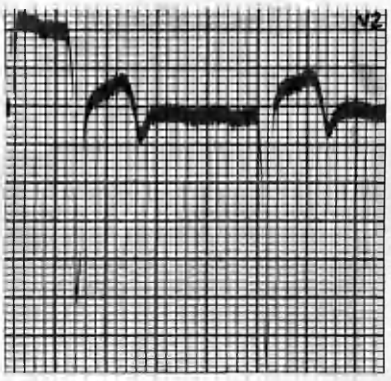


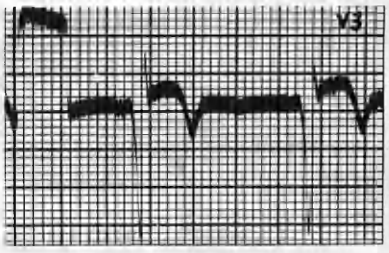
CASE 14,

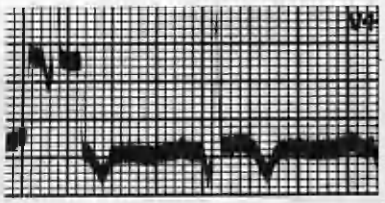
H.W. MALE, 55 YEARS.

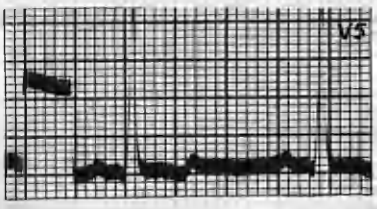
4 WEEKS
AFTER ONSET.

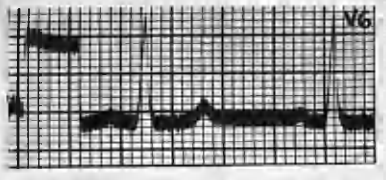


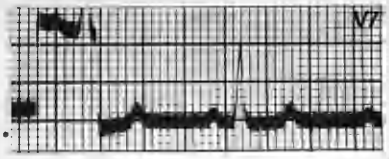












11 MONTHS
AFTER ONSET.

Case 15, D.H., male, 38 years.

This patient had sudden severe substernal pain awakening him out of sleep and lasting six hours a fortnight before his attendance at hospital. Blood pressure was 106/78. There was no cardiac enlargement but the sounds were of poor quality. The standard limb leads show no QRS abnormalities but the ST-T signs are very suggestive of anterior infarction - the cove - plane inversion of the T wave of lead I and the large upright symmetrical T waves of leads II and III. Lead VL has clearly derived its potentials from the central zone of infarction due either to clockwise rotation of the heart around its own long axis or to forward position of the apex. The praecordial leads are typical of an organising anteroseptal infarction. There are deep QS deflections in leads V2 and V3 with very deep inversion of the T waves. The deep QS deflection of lead V1 probably represents extension into the anterior part of the septum; it is unlikely to be a normal variant since the heart is not lying in the horizontal electrical position. The largely positive QRS of lead VF is indicative of an intermediate to vertical position. The apical lead V4 has a normal QRS complex but its ST-T formation is typical of the ischaemic zone around the

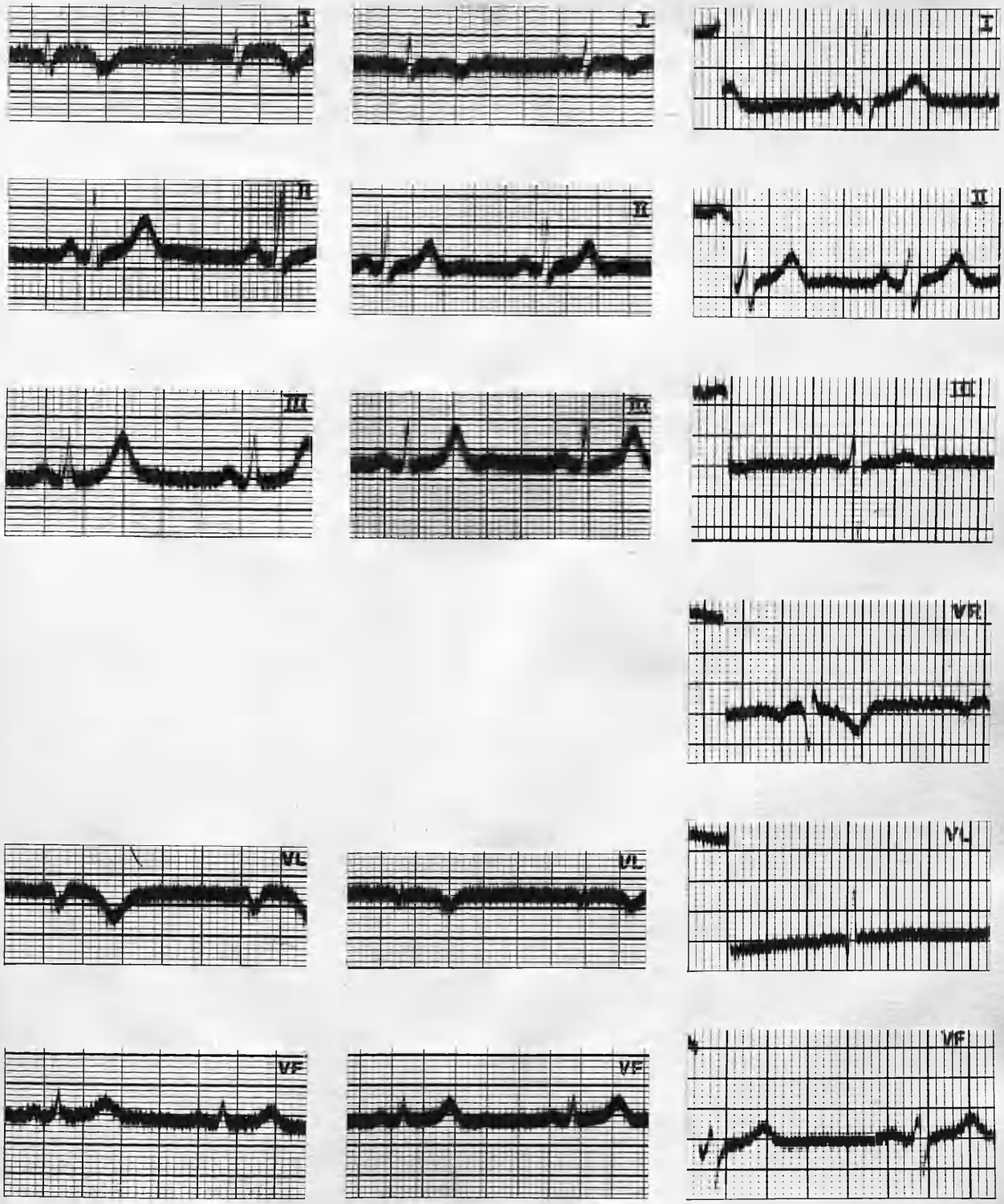
infarction.

An electrocardiogram taken ten weeks after onset shows little change in the standard limb leads. In lead VL however a small R wave has returned; in the previous electrocardiogram it was represented by a notch at the foot of the QS deflection. The praecordial leads show diminishing inversion of the T waves and in lead V3 the R wave has also reappeared, separating a prominent Q wave from a deep S wave and indicating some laminar recovery in the leftward edge of the infarction.

The position is further clarified by the tracing obtained more than two years after onset. The standard limb leads no longer show any sign of infarction but left axial deviation is clearly established. The unipolar praecordial leads supply a clue to the change of axis; lead VL shows a tall R wave, lead VF a RS complex, the S wave being the more conspicuous. This combination of signs is typical of a semihorizontal position. Presumably, healing of the infarct with local functional recovery of muscle accounts, in part, for the electrical axial shift but a sequence of changes, as striking as those displayed by leads VL and VF would favour the view that a minor change in anatomical axis had occurred, especially in view of the long interval of two years between the two tracings. The praecordial

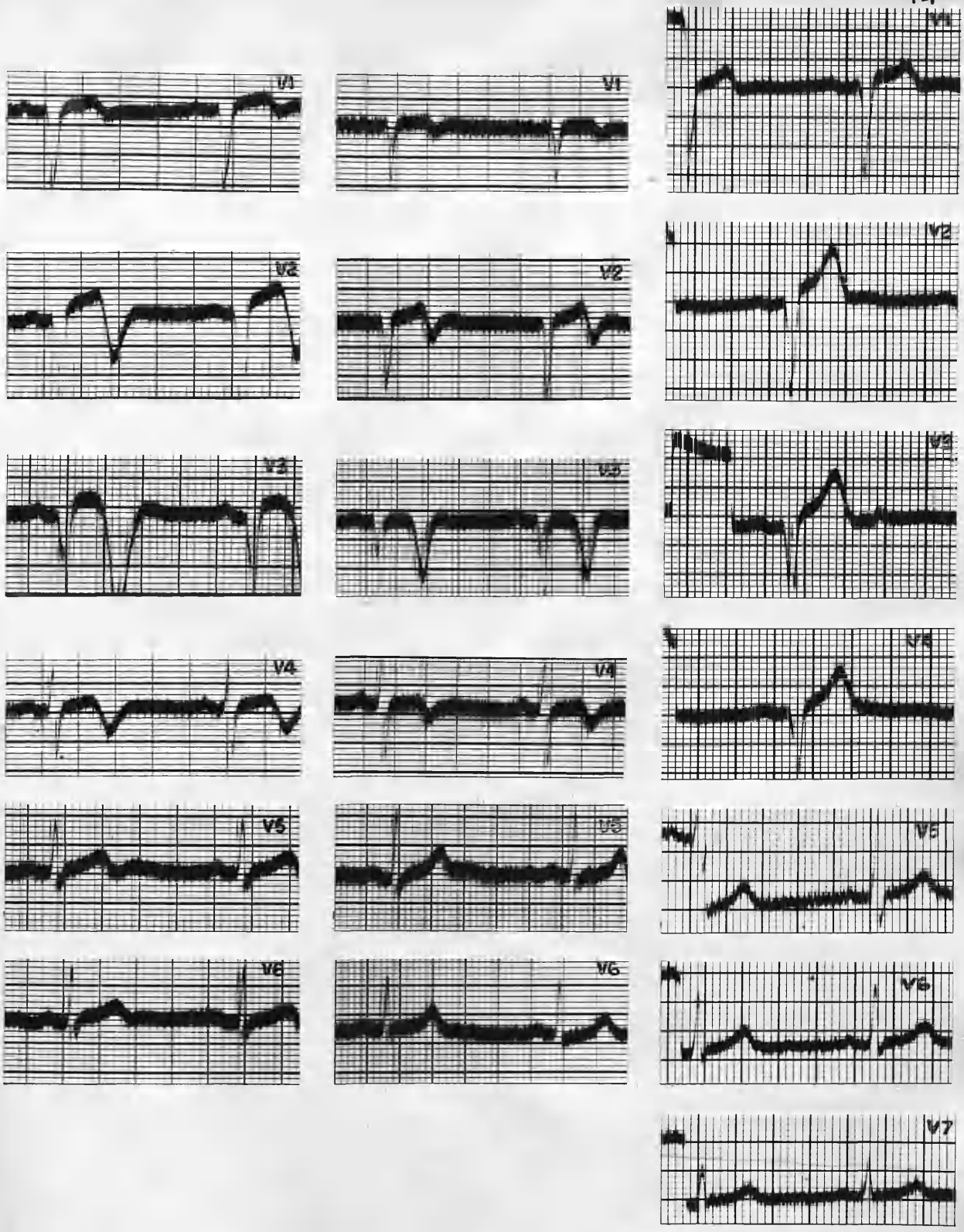
leads show persistent QS deflections in leads V1 and V2, the latter especially being the usual residuum of infarction; lead V3 shows the pattern of laminar recovery previously noted; lead V4 has a marginal pattern with a thick Q wave, measuring thirty per cent of the size of the subsequent R wave. Its pathological significance is emphasised by the absence of a Q wave in leads further to the left. In all the praecordial leads, the ST-T formation is normal.

This case is an example of a well healed anteroseptal infarction with change of electrical axis in a horizontal direction.



CASE 15, D.H., MALE, 38 YEARS.

16 DAYS	10 WEEKS	2 YEARS 3 MONTHS
AFTER ONSET.	AFTER ONSET.	AFTER ONSET.



CASE 15, D.H., MALE, 38 YEARS.

16 DAYS AFTER ONSET.

10 WEEKS AFTER ONSET.

2 YEARS 3 MONTHS AFTER ONSET.

A.

COMPARISON OF STANDARD LIMB LEADS AND UNIPOLAR LEADS IN THE DIAGNOSIS OF ANTEROSEPTAL INFARCTION

Myers et al (1948) studied twenty cases of anteroseptal infarction, correlating electrocardiographic and pathological findings. Eighteen of the twenty fail to show signs diagnostic of infarction in standard limb lead I. In at least six of the eighteen, the infarction was old and hence ST and T changes in any lead are minimal or unattributable to infarction and, in any case, the evidence of the previous anteroseptal infarction in these cases is confined to leads V1 to V3 or, perhaps, V4 at most. Furthermore, fourteen of the eighteen cases had left ventricular hypertrophy, the pattern of which, as explained above, is registered by lead I. All twenty cases came to autopsy but the cause of death in several cases was not the myocardial infarction. Renal abscess, gangrene of leg, peritonitis and uraemia occurred and it is possible that the electrocardiographic pattern of a focal lesion such as infarct was modified by the presence of a general toxaemic myocarditis. Similarly

/ Similarly, the effect of cedilanid in depressing the ST segment and thus obliterating any ST elevation cannot be ignored. The two cases of Myers' series which showed diagnostic signs in lead 1 were very recent cases and showed "a cardiac position which favoured transmission along a pathway from the C3 or C4 praecordial position to the left arm in place of the more common pathway from the axilla to the left arm".

Bain and Redfern (1948) in the course of a study on the Clinical Value of Unipolar Chest and Limb Leads describe ten cases of anteroseptal infarction in seven of which the T wave of lead VL is "just inverted". In only two of the seven however is the T wave inverted in standard limb lead 1 although in two others it is flat. No explanation is offered.

A short paper dealing with "The comparative value of the augmented unipolar limb leads versus the standard limb leads in myocardial infarction" was published by Fiske in 1950. He analysed twenty cases of infarction with postmortem confirmation. There were eight cases of anterior, seven of posterior and five of combined anterior and posterior infarction. He states that the Q wave of lead aVL is more valuable than that of standard limb lead 1 in the diagnosis of anterior infarction because of its greater dimensions not because of any greater specificity. This statement is subsequently qualified by the observation that lead aVL is more informative than lead 1 in /

/ in anteroseptal infarction and inferior to it in anterolateral infarction; the explanation is offered that in view of the actual position of the heart and the fact that the septum lies in an almost frontal plane, the septal effects are more readily projected to the left shoulder than are the lateral effects. The present work supports the facts in respect of anteroseptal infarction but not the explanation. However Fiske concludes by stating that the chest leads are superior to both the standard and the unipolar limb leads in the diagnosis of anterior infarction - a belief which is now generally held. He propounds the view that the R waves of the unipolar limb leads, in particular, of leads aVR and aVL, which contribute to the formation of the Q waves of the standard limb leads may be more than mere passive agents. He believes that the timing and size of these R waves may be altered by the presence of the infarction and thus increase the specificity of the Q waves of the standard limb leads, in particular of lead I and lead III respectively.

The present work lends favour to this thesis in respect of the more extensive anterolateral infarctions as discussed in a subsequent section. However, Fiske lays little emphasis on cardiac position as a determinant of the QRS patterns of the limb leads.

The ratio /

/ The ratio of the amplitude of T1 to T3 as an indication of a focal ischaemic myocardial lesion has been explored by Goldberger (1947) and by Dressler and Roesler (1948). Both studies are analyses of the pattern T1 less than T3, both being positive. Goldberger's material consisted of twelve-lead electrocardiograms from five hundred unassorted patients. He makes the statement succinctly that T1 is less than T3 when T of the left arm is negative. This may occur as a normal variant and also in the presence of left ventricular hypertrophy and of anterior infarction, both of which will be disclosed independently by adequate praecordial tracings. Goldberger does not discuss instances where the T wave of the right arm is more negative than that of the left and hence T1 is a low positive while the T3 of such cases is usually frankly positive. In general, he does not attach much diagnostic value to the ratio of T1 to T3. Dressler and Roesler studied sixty-six cases presenting the clinical picture of coronary disease (not specifically infarction) and also the pattern of T1 less than T3, both being positive. In fifty-six of them, myocardial infarction was diagnosed by chest leads; it was anteroseptal or anterolateral in fifty-five and a very early posterior lesion in the fifty-sixth case. They ascribed the T1 less than T3 pattern to the presence of the infarction. In fourteen of the sixty-six, this pattern was the only /

/ only electrocardiographic abnormality at the time of study but five of them had previously had QRS or T abnormalities so that the diagnosis of a focal myocardial lesion would not have been entirely missed. In nine of the cases the pattern of T1 less than T3 was, throughout the course of observation, the sole abnormality, but, probably, earlier and more numerous V leads would have disclosed more definite focal abnormalities. In none of the cases, for example, were unipolar limb leads taken. The authors point out that this pattern may occur in normal vertical hearts and in pulmonary emphysema. If these conditions are ruled out, the finding of T1 less than T3 "should strongly suggest an ischaemic myocardial lesion", which, if unrevealed by ordinary chest leads (sic) may lead to further exploration of the chest. This topic is again discussed in relation to the present series of anterior infarctions.

Of the present series of twenty-six anterior infarctions fifteen are anteroseptal in site and of the fifteen only one case shows normal standard limb leads in the presence of the signs of anteroseptal infarction in leads V2 and V3 one week after onset (case 1). This is not due to lead VL having derived its potentials from the intact axillary wall. On the contrary it shows signs derived from the marginal zone of the infarction, viz. a small Q wave, which is however significant in view of /

/ of the smallness of the R wave which follows it, very slight elevation of the ST segment and flat T wave. This transference of potentials from the marginal zone, although not apparently from the V positions, is facilitated by the vertical position of the heart seen by the tall R wave of lead VF. The deflections in lead VL are so small however that in lead I they have been obscured by the relatively large deflections of lead VR, the pattern of which, in reciprocal form, is largely that of lead I. Even the small Q wave of lead I is as much the reciprocal of the normal small R wave of lead VR as the transmitted minute Q wave of lead VL. During the second week after onset this infarct spread to involve the lateral wall. A similar spread of an anteroseptal infarction to form an anterolateral one was described in two cases by Rosenbaum et al (1945). Both patients exhibited the so-called premonitary symptoms of infarction and on that day electrocardiograms showed anteroseptal infarction. After two or three days' interval, symptoms returned in more severe form and electrocardiograms then showed anterolateral infarction. After the spread of infarction in the present case, lead VL becomes related to the central zone of infarction from which it now derives its potentials and the new changes are sufficiently pronounced, even when combined with lead VR, to cause an ST plateau and sharply inverted T wave in lead I, very

/

/ very suggestive of infarction. When the infarct has healed the standard limb leads are again normal although there is still a sharply inverted T wave in lead VL which is however annulled by the larger inverted T wave of lead VR in reciprocal.

A comparable relationship of leads, viz. lead VL in apposition to the central zone of an anteroseptal infarction is seen in case 2 ten days after onset. The tall R wave of lead VF indicates a vertical heart as in the preceding case. Lead VL has derived its pattern from the neighbourhood of the V3 position but its small QS deflection has been overshadowed in standard limb lead 1 by the large deflection from lead VR, the effect of which is to produce a tall R in lead I. The large, sharply inverted T wave in lead VL, notwithstanding the positivity of the reciprocal of the T wave of lead VR, is deep enough to effect a negative T wave in lead 1. The final form of lead 1 may be classed as very suggestive of anterior infarction.

Two further early cases, taken within two weeks of onset, also show signs in lead I very suggestive of infarction. Case 3 shows upward bowing of the ST segment and sharp inversion of the T wave but there is no Q wave and the R wave is tall. This pattern is closely similar to that of lead V5 in the ischaemic zone. The effect of lead VR in this case has been to increase the height /

/ height of R and the upward convexity of the ST segment. Recovery was rapid and one month after onset both the standard and the unipolar limb leads are normal while frankly abnormal signs persist in the anterior praecordial leads. Slight elevation of the ST segment with an almost flat T wave is seen in lead I of case 4 six days after onset. The R wave is small and there is a minute Q in lead I, the prominent Q of lead VL being minimised by the reciprocal of the short splintered QS deflection of lead VR. However, isolated ST-T abnormalities such as the above are not diagnostic, although very suggestive, of infarction. They also occur in acute pericarditis but their association with small R waves in lead I and with downward displacement of the ST segments in leads II and III is in favour of infarction.

A further example of isolated ST-T abnormalities in the standard limb leads is provided by case 5 where there is ST elevation in all three leads, greatest in lead II one week after onset. The absence of a Q wave in leads I and II is against the diagnosis of anterior infarction and the presence of a non-specific abnormal QRS complex in lead III is hardly sufficient to indicate posterior infarction. Elevation of the ST segment without associated QRS abnormalities also raises the question of pericarditis. As discussed in the case report, this patient had an anteroseptal infarction with involvement of the anterior /

/ anterior part of the septum and with subepicardial infarction of the lateral and posterior aspects of the apex. The electrical position of the heart was intermediate to horizontal with backward rotation of the apex so that lead VL as well as lead VR faced the posterobasal region of the left ventricle and recorded only those changes which were reciprocal to the infarction. Thus, while all three standard limb leads show ST-T changes derived by combinations of the ST-T abnormalities present in all three unipolar limb leads, the absence of significant QRS changes in the standard limb leads is due to the fact that the main part of the infarct with its significant QRS changes was limited to the anteroseptal wall and therefore diagnostic QR changes were confined to leads V1 to V3 and were therefore not transmissible to lead VL even if the left arm lead had occupied its usual relationship to the praecordial leads, viz. in apposition to the V5 position.

After the first two weeks following infarction lead I becomes decreasingly informative unless the signs of evolution and healing are delayed. Such circumstances account for the persistence of signs as late as three weeks after onset in case 6, and five weeks after onset in case 7. Both cases show doming of the ST segment and shallow inversion of the T wave, a pattern suggestive of a recent infarction, especially when associated with a small R wave as in both cases. Similar signs persisting till five weeks after onset are seen in case 8, which also shows depression /

/ depression of the ST segment in leads II and III. A similar ST pattern with cove-plane inversion of the T wave in lead I has apparently become fixed in case 9, where it is seen nine months after onset. Isolated abnormalities of the T wave are frequent in lead I in early and in late tracings. There is cove-plane inversion of T1 accompanied by unduly prominent and upright T waves in leads II and III in case 15 sixteen days after onset and similar though less striking T changes are seen four weeks after onset in case 14. Case 10 six weeks after onset and case 11 two and a half months after onset show cove-plane T waves in lead I, which would at least raise the possibility of previous anterior infarction and indicate the need for further electro--cardiographic investigation. Inverted T waves, although not of cove-plane shape, flat T waves and very low upright T waves, all of limited diagnostic value, are found in the later stages of healing. In subsequent months lead I may regain a normal outline although typical residual abnormalities persist in the anterior praecordial leads.

Thus, of the fifteen cases of anteroseptal infarction, twelve show ST-T signs in lead I which are suggestive of anterior infarction irrespective of its age. Only one case (No. 1) shows normal standard limb leads in the presence of a recent anteroseptal infarction. The remaining two cases (Nos. 12, 13) show isolated equivocal T changes in lead I but, in both cases, the tracings were not obtained until the second month after onset. It /

/ It has long been recognised that the left arm frequently derives its potentials from the V5 level and that it transmits this pattern to standard limb lead I after modification by the contribution of the right arm in reciprocal form. The transference of potentials from the V5 level to the left arm is facilitated by an intermediate to vertical position of the heart - a condition fulfilled by eight cases of the present series of fifteen. In acute anteroseptal infarction the only abnormality occurring in lead V5 is temporary inversion of the T wave and hence this is the only significant change which can ordinarily be expected in standard limb lead I. If a septal Q is present in lead V5, it will probably be recorded by standard limb lead I, where, however, it is unlikely to be misinterpreted because it is of short duration and the R wave following it, is tall. Among the cases of the present series which have repeated tracings, none shows a QRS formation in lead VR in which the direct influence of the anteroseptal infarction can be traced. (The change of pattern of the QRS of lead VR in case 4 is due to an axial shift). This absence of effect on the QRS deflection is not surprising in view of the fact that electrical changes arising in a localised area of the anteroseptal wall are at right angles to the frontal plane in which lead VR lies and therefore have little influence on it. A similar comment may be made in respect of the ST-T formation with this addition however : changes /

/ changes affecting the T wave are acknowledged to be more widespread than those involving the QRS since they arise from the zone of ischaemia surrounding the infarction. Hence the chances are greater that lead VR will appose the zone of ischaemia rather than the central zone. This relationship has occurred in two cases of the present series; in case 5, lead VR faces the back of the heart and shows an upwardly concave ST segment and an upwardly peaked T wave, clearly the reciprocal effects of the infarction referred to the right arm because of an unusual cardiac position. In theory these features should have exaggerated those of lead VL in the formation of lead I but lead VL in this case also faces the back of the heart and the result in lead I is a cancelling out of the ST-T signs. The other case, No. 9, shows symmetrical upward peaking of the T wave in lead VR, the reciprocal of the large inverted T waves in the apical and anterior axillary leads. In this case T of VR in reciprocal exaggerates the inversion of T of VL in the formation of lead I. In these two instances lead VR contributes specific ST-T effects derived from the infarction in the formation of standard limb lead I.

Frequently however the effect of lead VR is to decrease the size of the deflections of lead VL so that standard limb lead I is of low voltage throughout and is therefore of less value in diagnosis. For example, if lead V5 and the left arm lead show sharply inverted /

/ inverted T waves, which is not uncommon in anteroseptal infarction, and if the right arm lead also shows an inverted T wave, which is a normal variant, the effect in standard limb lead I is the registration of a flat or shallow inverted T wave. In early cases it is rare for the normal inverted T wave of lead VR to be deeper than the sharply inverted T wave of lead VL and thus for the T wave of lead I to be upright but this is not uncommon in healed lesions in the second six months or in the second year after onset (cases 1, 7, 11, 12, 15). Occasionally as stated above, a spurious Q wave may arise in standard limb lead I if a normal early R wave occurs in lead VR and if its reciprocal effects are not cancelled by a simultaneous upright deflection in lead VL (case 1).

The ratio of the T wave of standard limb lead I to that of lead III, both being upright, has been noted in the present series of fifteen anteroseptal infarctions. TI is less than TIII (but both are positive), in one or sometimes in two electrocardiograms of seven cases (1, 6, 7, 10, 11, 12, 13). It can be ascribed to the presence of the infarct in six cases. In the seventh case (case 6) it may be due entirely to the vertical position of the heart; the tracing in question was taken one year and three months after onset by which time the residual signs of infarction were confined to the anterior praecordial leads. Of the six cases, one (No. 1) shows the pattern /

/ pattern of T1 less than T3 four hours and also one week after onset and again after seven and a half months. The occurrence of the pattern in both the earliest stage of infarction and again as a late sign was noted by Dressler and Roesler (1948). On the other hand, two of the cases (7, 12), in a further late tracing, at two years and two months and one year and eight months after infarction, respectively, show higher T waves in lead I than in lead III although both show typical residual signs of infarction in the anterior praecordial leads. The experience with the above seven cases does not favour the view that the T1/T3 ratio is of special diagnostic value.

Sometimes the axis of the heart is such that the potential variations of the left arm are derived not from the axillary leads but from the V3 and V4 praecordial positions or from points slightly above this level. This reference of potentials requires either forward rotation of the apex or some degree of clockwise rotation of the heart round its own long axis - a movement more commonly associated with vertical than with horizontal hearts. In these circumstances in the presence of anteroseptal infarction lead VL may record a significant QR deflection, elevation of the ST-T junction, upward displacement of the ST segment and, subsequently, inversion of the T wave. These findings are transmitted, although modified by those of lead VR to standard limb lead I. Unfortunately /

/ Unfortunately if the deflections of lead VL are small, they may be entirely masked in lead I by the pattern of lead VR, especially if the latter shows large deflections of opposite algebraic sign, in reciprocal, to those of lead VL.

Informative signs in standard limb leads II and III are very scanty in anteroseptal infarction. When there is upward displacement of the ST segment in lead I there may be downward displacement of the segment in leads II and III, especially the latter, derived from the pattern in lead VF (case 9). Such a downward ST displacement is typical of the intact left ventricular wall opposite an acute transmural or subepicardial infarction. More frequently, lead VF shows a large symmetrical and upright T wave which is a reciprocal effect derived from the zone of ischaemia (cases 1, 2, 7, 10 and 15). A vertical or semivertical cardiac position favours this transference of potentials.

With the passing of the stage of injury and the resolution of ischaemia all abnormalities of the ST segment and T wave tend to disappear. With further healing the shortened R waves of the praecordial leads may become tall again. These favourable signs tend to appear first in leads over the periphery of the infarct, viz. in leads V5 and in V4, in that order; hence lead VL and therefore lead I may have returned to normal while there are still frankly /

/ frankly pathological signs in leads over the centre of the infarct, say in leads V3 and V2. It is therefore increasingly improbable that lead I will show any abnormalities after the first few weeks if resolution and healing are progressing satisfactorily and if the heart is in its usual position with reference to the left arm, i.e. with the lateral ventricular wall apposed to it. Nevertheless fixation of T wave pattern in lead I frequently occurs before the normal form is regained and although a shallow inverted T wave or a flat T wave in lead I is not necessarily indicative of a previous anterior infarction, their presence at least calls for further electrocardiographic investigation.

Another factor contributing to the registration of diagnostic signs in lead VL and in lead I in the present series is that in all cases the illness was the first of a cardiovascular nature experienced by the patient and that therefore the pattern of infarct was superimposed on a presumably normal electrcardiogram. None of the cases in the first instance shows left ventricular hypertrophy or dilatation, clinically or electrocardiographically. It is well known that this condition is a frequent cause of the failure of lead VL, and therefore of lead I, to record the signs diagnostic of anteroseptal infarction since the enlarged left ventricle causes the heart to assume a horizontal position with some degree of /

/ of counterclockwise rotation, and hence the intact lateral wall of the left ventricle is presented to the left arm. Similarly none of the cases presents signs of diffuse myocardial degeneration and dilatation which also tend to cause a horizontal cardiac position.

SUMMARY

Of fifteen cases with electrocardiograms diagnostic of anteroseptal infarction an abnormal ST-T pattern is seen in standard limb lead I in thirteen, which, while not indicative of infarction, is suggestive of it and points to the necessity of further electrocardiographic examination by praecordial leads.

The earlier the tracing the more likely are diagnostic signs to be found in standard limb lead I.

Intermediate to vertical electrical positions are those most favourable for the transference of signs from the marginal or central zones of the infarct to lead VL and thence to standard limb lead I. Severe backward rotation of the cardiac apex is a deterrent to the transference of the signs to lead VL.

The contribution of lead VR to the formation of standard limb lead I is variable. Most commonly it /

/ it obscures or mitigates the signs of infarction in lead VL; occasionally it supplies a spurious Q wave; occasionally its ST-T formation is specifically, although reciprocally, affected by the infarction and hence it tends to augment any ST-T signs in lead VL which are derived directly from the infarction.

The pattern of T1 less than T3, both being positive, is an unreliable sign of anteroseptal infarction.

The absence of other cardiac lesions, in particular of left ventricular hypertrophy, is a favourable factor for the registration of diagnostic signs in standard limb lead I.

B.

UNIPOLAR ELECTROCARDIOGRAPHY AS AN AID TO DETERMINING THE SITE, EXTENT and HEALING of ANTEROSEPTAL INFARCTION

The previous pages have demonstrated that the unipolar praecordial leads are essential for the diagnosis of anteroseptal infarction. But apart from diagnosis as such, they also indicate the site of the lesion and, to some extent, its size. In the communication previously cited, Myers et al (1948) studied twenty cases of anteroseptal infarction all with postmortem examination. Since the final diagnosis rested more on the pathological than on the electrocardiographic signs, their tracings include variations from the classical pattern described above as well as typical examples. Nine of their twenty cases showed QS deflections in V2, V3 and/or V4 although in two of the cases the QS complex was restricted to one lead. A tiny Q wave followed by an rS deflection in lead V1 was interpreted as indicating septal extension of the infarct which was confirmed at postmortem examination. A further seven of the cases showed a QS deflection in lead V1 as well as in other anterior praecordial leads; in six involvement of the septum was found and was presumably the cause of the absence of the small initial R wave in these cases. The explanation of the QS wave in lead V1

/ lead V1 of the seventh case is not clear; it is not explained by horizontal cardiac position for the heart was vertical in this case. Nevertheless in six other cases in all of which a small initial R wave was present in lead V1, there was at post-mortem examination, involvement of the septum, but, as Myers emphasises, it was patchy and surviving muscle or activation of the free wall of the right ventricle may have accounted for the preservation of the R wave in lead V1. Two of the original twenty cases retained an R wave in all the anterior praecordial leads without the development of a Q wave. In the first, the infarct was small and intramural and in the other, it was patchy; the preservation of both subendocardial and subepicardial layers may account for the absence of QRS abnormalities. A close correlation was found between diagnostic QRS abnormalities in leads V3 and V4 and infarction of the apical one-third to two-thirds of the anteroseptal wall. In two cases with apical infarction lead V4 presented no diagnostic signs - a lack of correlation which is explained by a cardiac position favouring "transmission of potential variations of the apex to a point medial to the mid-clavicular line". Similarly borderline QR complexes may occur in leads V5 and V6 as a result of clockwise rotation of the heart about its own long axis and about an anteroposterior axis. Sometimes it may be impossible to say whether QS complexes /

/ QS complexes in the first two or three praecordial leads are attributable to the septal extension of an infarct or to involvement of the basal third of the anteroseptal wall.

In the course of a paper on the electrocardiographic patterns in "slight coronary attacks" Papp and Smith (1951) found an anteroseptal localisation in seventeen cases out of a total of thirty-three cases. The R wave was small or absent in three records, all showing anteroseptal ischaemia and pathological Q waves were encountered in three instances of the anteroseptal pattern. Abnormalities of the ST segment and T waves were prominent in all their cases. The tracings of five of their anteroseptal infarctions are reproduced. However, the number of praecordial tracings is inadequate in number; one case has only a V4 tracing for its first record and the other four cases have only three or four chest leads; positions V1 and V3 or CR2 and CR3 or CR1 and CR3 or V1 and V2 have been omitted. The present writer would submit that had full chest tracings been available, more abnormalities of the QRS complex would have been discovered and the diagnosis of anteroseptal infarction, rather than anteroseptal ischaemia would have been justifiable in several cases. Their case 3 bears a strong resemblance to case 3 of the present series which shows the signs of a septal and anteroseptal infarct and similarly their case 5 is very /

/ very similar to the present case 11 which also shows septal and anteroseptal infarction. They emphasize that normal or near-normal electrocardiograms may be obtained after very variable intervals. Their case 1 three months after onset is said to show a normal record, but there is notching of the upstroke of the regenerated R of leads V2 and V3 and the latter has a double T wave and also there is a delayed R wave in lead VL with inverted T, which, when combined with the normal inverted T in lead VR, gives a flat T wave in standard limb lead I. These are surely the stigmata of a well healed anteroseptal infarction. It is difficult to discuss the late signs in the other published tracings since certain V or C positions have been omitted.

All fifteen cases of anteroseptal infarction described in the present work have QS deflections in one or more of the anterior praecordial leads irrespective of the age of the infarct. Thirteen have electrocardiograms taken in the early weeks of illness, of whom ten show a central zonal pattern, i.e. a deep QS deflection in leads V2 and V3 - a distribution which is the classical pattern of anteroseptal infarction. (cases 1, 4, 5, 7, 8, 10, 12, 13, 14, 15). Of the remaining three, one has a deep QS deflection in lead V2 only but a significant QR in lead V3 (case 3); the second has a deep QS in lead V2 only and a normal RS in lead V3 (case 6); and the third has a deep QS deflection confined to lead V3 (case 2). Lead V4 shows much variation /

/ variation. It may show a marginal zonal pattern, i.e. a significant Q wave and a small R wave, when a central zonal pattern is seen in lead V3 (cases 1, 10, 13, 14). In one case (No. 4) lead V4 as well as leads V2 and V3 shows a deep QS deflection suggesting that the apex as well as the anteroseptal wall is involved transmurally. There is one instance (case 6) of non-specific abnormality in lead V4, viz. a broad notched S wave but no Q wave. Seven cases show normal QRS complexes in lead V4. (Nos. 2, 3, 5, 7, 9, 11, 15). All have prominent S waves, i.e. the ventricular pattern is transitional in type, marking the level at which the right ventricular pattern changes to the left ventricular pattern. As a rule, this takes place in lead V3 in a normal heart but in the present seven cases its displacement to lead V4 is probably no more than a normal variant present before infarction. The ST-T deflections of leads V2, V3 and V4 conform to the typical pattern. Elevation of the ST junction and convexity upwards of the ST segment are greatest, as a rule, in lead V3 whereas the subsequent inversion of the T wave is usually deepest in lead V4 where it may reach fifteen millimetres (cases 1, 11).

Twelve of the thirteen cases with early tracings have normal QRS complexes in leads V5 and V6. Case 4 which has a deep QS complex in lead V4 as well as in leads V2 and V3 shows a small RS deflection in lead V5. The

/ The absence of a q wave in this lead would suggest that the lateral aspect of the apex had escaped infarction, at least in its subendocardial layers, and is the reason for excluding this case from the group of anterolateral infarctions although it must be admitted that the criteria for separating anteroseptal and anterolateral infarctions are at times arbitrary. In spite of normal QRS deflections, leads V5 and V6 usually display abnormal ST-T signs, viz. upward bowing of the ST segment and inversion of the T wave. Taken in conjunction with the ST-T contour of lead V4, these signs are generally regarded as indicating an underlying zone of ischaemia especially if they are more conspicuous in lead V5 than in lead V6 but the possibility of their arising from pericarditis cannot be ignored. Furthermore, in later tracings, inversion of the T waves of leads V5 and V6 may be due to left ventricular hypertrophy, especially if associated with tall and slightly delayed R waves. (cases 6, 9, 10).

Of the thirteen cases with early tracings only two show the classical pattern of anteroseptal infarction in lead V1 and V2, viz. the retention of a small initial upright deflection in lead V1 and its disappearance or diminution in lead V2. (cases 2, 10). This small R wave of lead V1 is derived from activation of the left side of the septum and its preservation indicates that the septum is intact. The remaining eleven cases have a QS deflection in lead V1 as

/ as well as in lead V2. The decision as to whether the absence of a small R wave is due to infarction or to other causes, e.g. horizontal position, depends on the QRS pattern in lead V3, and the ST-T contour in the first four praecordial leads. Ten of the eleven cases have diagnostic QS or QR complexes in lead V3 along with classical ST-T signs in the first four praecordial leads so that the QS deflection of lead V1 may rightly be attributed to infarction of the septum. In the remaining case, (No. 3) QS deflections are confined to leads V1 and V2 but there are ST-T changes typical of recent infarction in the first five praecordial leads so that the diagnosis of septal involvement is justified. The ST-T changes in lead V1 are much less constant than in leads further to the left. Very slight elevation of the ST junction and inversion of the end of the T wave are the signs most frequently observed in this lead.

Of the remaining two cases of the original series of fifteen, neither of which have early tracings, the first (No. 9) shows persistent QS deflections in leads V1, V2 and V3 as late as nine months after onset but by that time the ST-T formation of these leads has returned to normal. The other (No. 11) shows signs suggestive of septal infarction in the first tracing two and a half months after onset, but at sixteen months after, there are deep QS deflections in leads V1, V2 and V3 with normal /

/ normal ST-T waves typical of a previous septal and anteroseptal infarction.

Compared to the praecordial leads, the unipolar limb leads show a much greater diversity of pattern. If the heart is vertical there is very little alteration in the deep negative QS deflection which lead VL and also lead VR normally exhibit in such instances (cases 6, 10). In other positions of the heart the height of the R wave of lead VL is subject to much variation due largely to cardiac position. The heart in case 3, for example, is in the semihorizontal position and lead VL shows a conspicuous R wave whereas in case 13, the heart is intermediate and shows a small RS deflection in lead VL. Irrespective of the height of the R wave, the ST segment may display upward bowing and the T wave may be inverted; if the preceding R wave is short, the possibility of involvement of the subepicardial layers of the lateral wall cannot be excluded. Such ST-T formations considered alone suggest derivation from the ischaemic zone surrounding the infarction.

The classical picture of anteroseptal infarction does not include a significant Q wave in lead VL as does that of anterolateral infarction. However five cases have been classed as anteroseptal although they do show central or marginal zonal patterns in lead VL. In one (case 11) the Q wave of lead VL although 33% of the tall R wave /

/ R wave following it, is probably "non-significant", being derived from intact posterior portions of the septum, as explained in the text. In another (case 4) the infarction was specially large and but for the absence of a Q wave in leads V5 and V6 might have been classed as anterolateral rather than anteroseptal. The remaining three cases show a cardiac position specially favourable for the transference of a marginal or even a central zonal pattern to the left arm. In case 1 the heart is semivertical with forward rotation of the apex; in the second week of illness this infarction did spread to the lateral wall and the marginal zonal pattern of lead VL became replaced by a central zonal pattern. Closely similar is case 2 where, in a vertical position with forward rotation of the apex, a central zonal pattern is recorded by lead VL, although the praecordial pattern is typically that of anteroseptal infarction, and, lastly, case 15, where the heart is first in the intermediate to vertical position; lead VL then registers a central zonal pattern but when it rotates into a semihorizontal or horizontal position it shows a marginal pattern. Thus caution must be exercised in interpreting QS or QR deflections in lead VL, since, in intermediate to vertical hearts with or without forward rotation of the apex, they probably do not indicate involvement of the lateral wall.

As discussed in the previous section, lead VR as a

/ as a rule, presents no specific abnormality attributable to the infarction. Occasionally sharp upright T waves may occur in lead VR and are typical of the unaffected wall opposite an infarction (cases 1, 2, 7).

Similarly with regard to lead VF there are as a rule no specific abnormalities directly attributable to anteroseptal infarction. However case 5 is of particular interest in that the praecordial leads show the typical pattern of anteroseptal infarction whereas lead VF shows very small Q and R waves with ST plateauing, i.e., a marginal zonal pattern. Leads VR and VL clearly face the back of the heart and their ST-T patterns are typical of the reciprocal effect of infarction. There has therefore been backward rotation of the apex in this case so that the edge of the infarction is in relation to the diaphragm while lead VL faces the unaffected posterior or posterolateral wall. Hence in this cardiac position a marginal zonal pattern in lead VF does not necessarily mean extension of infarction to the posterior part of the apex.

The regressive changes following infarction show much variation in the time of their occurrence in the unipolar leads.

The ST-T changes in the apical and axillary leads may disappear (cases 1, 7, 11, 12), or may persist for many months (cases 4, 8, 9). They may ultimately present /

/ present the shelving ST-T contour associated with left ventricular hypertrophy (cases 6 and 10). If T inversion persists, it is usually deepest in leads V3 and V4 as in the earlier weeks. Regression of the ST-T signs may ultimately be complete in leads over the right side of the praecordium especially in tracings taken a year after onset. The most persistent signs are the deep QS deflections which are as a rule permanent in the leads which originally showed them. In two cases there are signs which would suggest some measure of recovery in the septal extension of the infarct. A minute initial upward deflection returns to lead V1 of case 14 and an early R wave is clearly seen in leads V1 and V2 of case 3 within a month after onset where, previously, there was no R wave. In this case the pathological process which originally involved the septum and caused the disappearance of the original R wave has been of a temporary, reversible nature, probably oedema or congestion.

Signs of recovery are not infrequent at the leftward margin of the infarct, especially in lead V4 and later in lead V3. Q waves tend to become shorter and R waves taller. A notch in the deep QS of lead V3 may become, in later months, an R wave, its peak just reaching above the iso-electric level and a smooth-limbed QS deflection in lead V2 in the early weeks may later develop a notch /

/ notch as an indication of some scanty laminar recovery. Cases 1 and 15 are excellent examples of this process. In the case cited above (No. 3), where the early septal R wave returns, there is, concurrently, increase in height of a late R wave in lead V3 indicating functional recovery in the left margin of the infarct also. The notching of the QS in leads V1 and V2 is probably the analogue of this R wave in lead V3. Sometimes the laminar nature of the process of recovery is well seen in the leftward margin of the infarct. The original R wave of lead V4 may persist more or less unchanged or may become taller in which case it may also re-emerge in lead V3.

On the other hand, if the basic praecordial pattern shows a sharp transition from a right to a left ventricular form, so that there is no S wave in or after lead V4, the same processes of repair and regeneration cause another distinctive pattern quite unlike that described above. Simultaneously with signs of increased left ventricular activity, viz. increased height of the R waves and downward shelving of the RS-T segment in the axillary leads, a new late R wave develops where previously there was none; in lead V3 in case 14 and lead V4 in case 10; in both cases there are persistent RS-T abnormalities. Such a pattern tends to occur in vertical to intermediate hearts with some degree of counterclockwise rotation. (cases 10 and 14).

/ Furthermore, regenerative processes may in themselves be responsible for a change in electrical axis. In case 10 it is probable that there was an increase in the counter-:clockwise rotation of the heart around its own long axis in later months, because lead VL develops a pattern usually derived from the posterior wall of the heart and a pattern previously exclusive to lead V1 is now shared in large measure by lead V2. There are two other examples of change of electrical axis, (cases 4 and 15), in both instances around an anteroposterior axis. At first the heart is in an intermediate to vertical position, out of which it rotates into a semihorizontal or horizontal position. In case 4 the change is seen as early as seven weeks after onset when left axial deviation has become clearly established; in addition, there is backward rotation of the apex for the right arm registers, for the first time, a pattern derived from the back of the heart. It will be recalled that the infarction in this case was specially large with possible involvement of the subendothelial muscle in the lateral wall. In addition, healing is only partial as seen in the praecordial leads and signs of a local conduction defect develop in lead VL. The presence of a fairly large, incompletely healed infarction in the anteroseptal wall with probable lateral extension may well have caused the heart to assume the above position. In the second example (case 15), the long interval of two years elapses between the two /

/ two relevant electrocardiograms and the sequence of changes displayed by the left arm and left leg leads is striking enough to suggest that not only did local functional recovery occur in certain portions of the infarction but also that a minor shift towards the horizontal occurred in the anatomical axis. The latter movement however may be partially attributable to physiological changes in the heart's spatial relationships due to age.

SUMMARY

The unipolar praecordial and limb lead electrocardiograms of fifteen cases of anteroseptal infarction have been analysed. All of them have QS deflections in leads V2 or/and V3 irrespective of the age of the infarction. In the thirteen cases which have early tracings, the ST-T deflections of the anterior praecordial leads are typical of infarction; in older tracings deep QS deflections persist but the ST-T formation returns to normal in these leads.

Leads V5 and V6 have usually a normal QRS deflection but they may show upward bowing of the ST segment and inversion of the T wave derived from the zone of ischaemia.

A small initial upright deflection or R wave is seen in leads V1 and V2 in two cases only. The reasons for /

/ for considering that the absence of the R wave from leads V1 and V2 is caused by involvement of the septum are discussed.

Occasionally marginal or even central zonal patterns may be transmitted to lead VL but, as a rule, inversion of the T wave is the sole abnormality. Rarely a marginal zonal pattern is recorded by lead VF.

In one case the unipolar praecordial leads demonstrate spread of infarction from anteroseptal to anterolateral sites.

Signs of repair and regeneration may be demonstrable in the praecordial leads, viz. the return of the small early R wave in leads V1 and V2, an increase in height of the R wave of the apical and axillary leads, the development of a late R wave in leads V4 and subsequently in V3. Regression of ST-T signs may be complete.

In late tracings the signs in the axillary leads may suggest early left ventricular hypertrophy.

The importance of electrical cardiac position is stressed as a determinant of the formation of the unipolar leads and of the patterns of the regenerative processes.

Change of electrical axis may occur in the course of the healing of infarctions. This may occur in association with satisfactory healing and good functional recovery or in the presence of inadequate healing, and possible myocardial dilatation. A change of anatomical axis may also arise.

SECTION IV

ANTEROLATERAL INFARCTION

SECTION IV

ANTEROLATERAL INFARCTION

INTRODUCTION

The signs that a myocardial infarction is involving the lateral aspect of the apex as well as the anterior cardiac wall have been clearly set out by Myers et al (1948), whose criteria may be summarised as follows. Diagnostic QRS abnormalities are found in leads V5, V6 and/or VL as well as in one or more of the first four praecordial leads. The pattern in leads V1 to V4 is, in general, comparable to that produced by an infarct confined to the anteroseptal wall, viz. by a QS deflection in leads V3 and V4 and sometimes also in V2. A similar QS deflection in lead V1 may indicate extension of the infarct into the septum. An abnormal QS or QR complex may be found in all three leads, V5, V6 and VL; less commonly the QS or QR is limited to leads V5 and V6 or to leads V5 and VL.

Wilson (1944) pointed out that the lateral wall of the heart is the chief determinant of the potential variations of the left arm in most positions of the heart except the vertical; hence lead VL shows, in an anterolateral infarction, an abnormal QR or, less commonly, QS pattern with ST-T sequential changes, which are transmitted to standard limb lead I after modification by the reciprocal

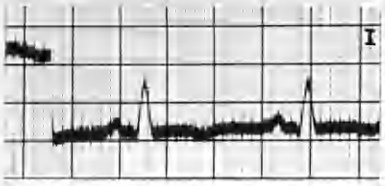
/ reciprocal of lead VR. However, the pattern of lead VR is subject to much physiological variation, according to the region of the heart which it faces (Goldberger, 1947). When lead VR has its usual relationship to the right ventricle, viz. in apposition to its anterior wall or cavity, it shows a small or minute R wave, usually designated 'r' wave, followed by a deep S wave and an isoelectric ST segment. The r wave may be absent if the right arm lead faces the ventricular cavity but is not in apposition to the septum. If lead VR faces the anterior wall of the right ventricle its T wave is upright; if it faces the cavity, its T wave is inverted. These patterns occur in vertical, intermediate or horizontal hearts with forward rotation of the apex or with mean position of the apex. If, however, there is backward rotation of the apex or marked clockwise rotation of a vertical heart around its own long axis, lead VR faces the posterior wall of the heart and shows prominent Q and R waves and downward T waves. In either group the anterolateral infarction may be extensive enough to involve the anterior part of the septum and adjacent portion of the right ventricle and thus a fairly direct relationship is established between the right arm lead and the infarction. Thus the contribution of lead VR in the formation of standard limb lead I is partly physiological and partly pathological in origin, as is illustrated by the following reports of eleven cases of anterolateral infarction.

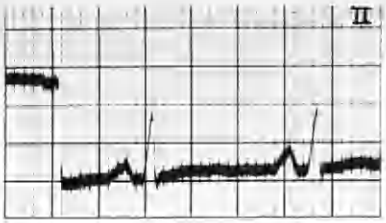
Case 16, J.D., male, 52 years.

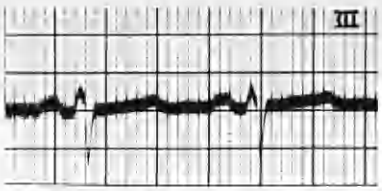
This patient, a dock labourer, gave a history of dyspnoea on exertion of at least six months duration. Three weeks before admission to hospital he had had sudden and severe praecordial pain lasting several hours and requiring morphine for its relief. A second attack of pain occurred three days before admission. During the first week of his hospital residence pericardial friction was audible on one occasion and on another there was a paroxysm of auricular fibrillation, the blood pressure falling from an admission level of 176/110 to 145/95; otherwise the cardiac sounds were invariably soft and there were usually signs of bronchitis. Radiological examination showed slight increase in the transverse cardiac diameter and slight congestive changes in the lung fields.

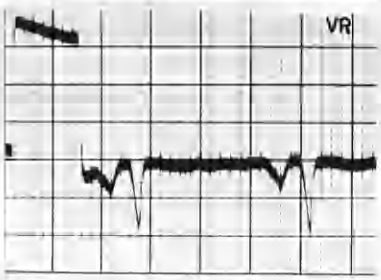
A thirteen-lead electrocardiogram obtained four weeks after onset shows broadening of the QRS complex in all leads. There is shallow inversion of the T waves in lead I and practically flat T waves in lead II. The standard limb leads do not suggest a recent acute infarction but they would certainly raise the possibility of an anterior coronary insufficiency. The unipolar limb leads with the broad short Q wave in lead VL and the delayed intrinsic deflection of leads VL and VF suggest a conduction defect in the lateral

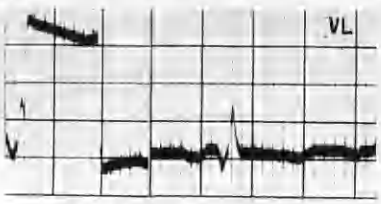
and posterolateral walls of the left ventricle. The ST-T deflection of lead VL is consistent with a local conduction defect; it does not suggest a recent acute anterolateral infarction. It should be noted that the prominent Q wave of lead VL has been almost obliterated in lead I by the reciprocal of the broad deep QS deflection of lead VR. Praecordial tracings V1 and V2 show the deep S waves commonly associated with left ventricular hypertrophy but otherwise these leads are normal. There is slight elevation of the ST-T take-off in lead V3 but otherwise it also is normal. The minute R wave of leads V1, V2 and V3 is no longer discernible in lead V4, the ventricular complex of which is represented by deep Q and late R waves. There is frank elevation of the ST segment here with early inversion of the T wave. This ST-T abnormality is again seen in lead V5 and would indicate recent focal infarction. As before the broad short Q wave of lead V5 with delayed R wave indicates a local conduction defect. In this case the infarction is chiefly apical and lateral. The form of the QR deflections of leads V4 and V5 is very suggestive of an older lesion with some laminar recovery but the ST-T changes are more likely to be due to a recent acute lesion. This interpretation is in accordance with the clinical history although dyspnoea and not pain had been the chief feature previous to the recent illness.

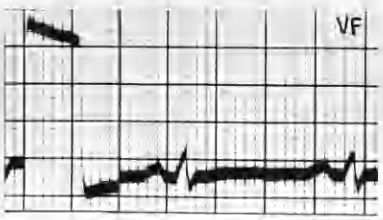


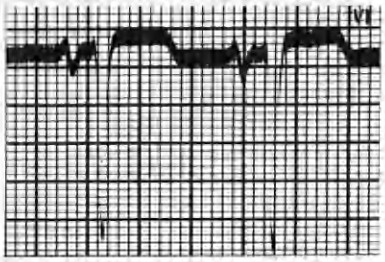


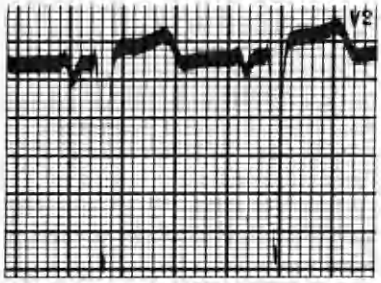


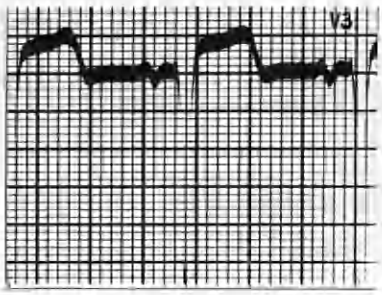


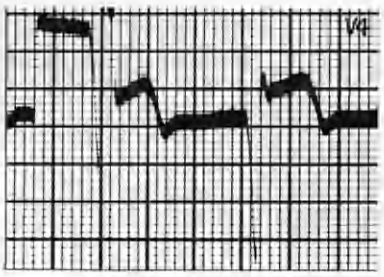


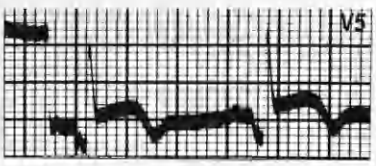


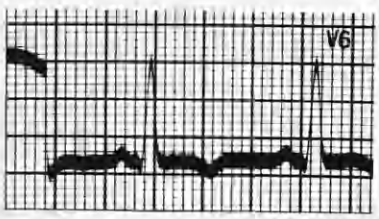


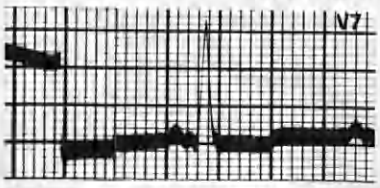












CASE 16, J.D. MALE, 52 YEARS.

4 WEEKS AFTER ONSET.

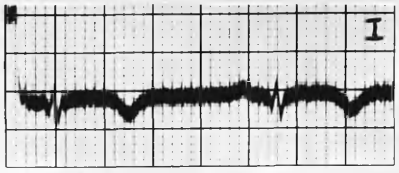
Case 17, H.H., male, 48 years.

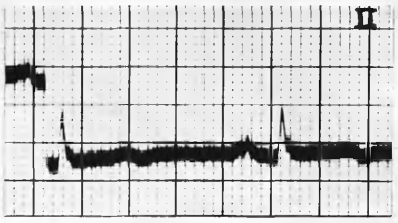
This patient had had dyspnoea on erection and occasionally substernal tightness for several months prior to the onset of his acute illness, which began with an attack of intense burning pain in the lower sternal area radiating into left or right axilla. This attack lasted one hour. Subsequently he had recurrent attacks of similar pain, usually when he exerted himself.

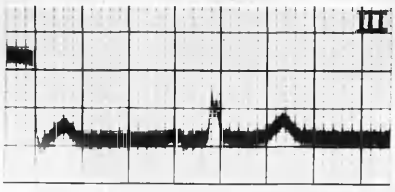
Patient was a heavily built man without cyanosis or dyspnoea. B.P. 140/90; 130/70; 110/70. The cardiac apex-beat was not palpable. The sounds were usually of average intensity. The chest was invariably clear.

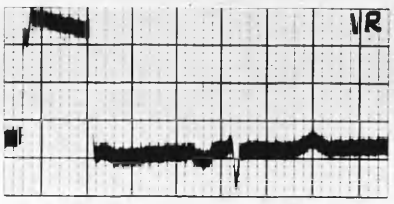
An electrocardiogram obtained nine weeks after onset of pain shows the typical signs of an anterior infarction in lead I; there is also slight upward convexity of the ST segment in lead II. Lead VR with its rS complex and upright T wave is clearly facing the anterior surface of the right ventricle whereas the prominent R wave of lead VF is derived from the left ventricle and together they indicate a vertical or semi-vertical heart with forward rotation of the apex. This is further substantiated by the central zonal pattern of lead VL instead of the marginal pattern usually seen in this lead. When modified by the reciprocal of lead VR however, the late QS deflection

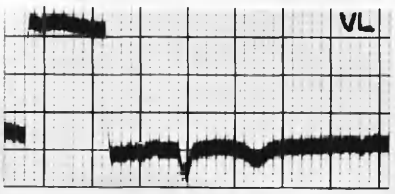
of lead VL provides chiefly the S wave of standard limb lead I. The early Q wave of lead I is provided by the reciprocal of the early R wave of lead VR. The praecordial leads are typical of an anterolateral infarction. The retention of the small initial upright deflection in leads V1 and V2 indicates that the left side of the septum has escaped. The prominent QS deflection in leads V3 and V4 is typical of the central zone of infarction. Lead V5 shows a marginal pattern with a Q wave at least 25% of the succeeding R wave, and lasting almost 0.04 sec. The inversion of the T waves indicates an organising lesion; the deepest inversion is in lead V4 which is also a typical finding. The particular interest of this case lies in the configuration of lead VL with its central zonal pattern.

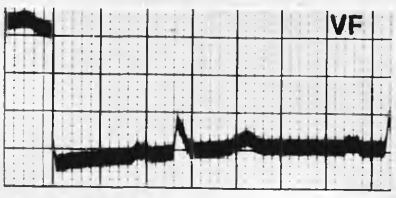








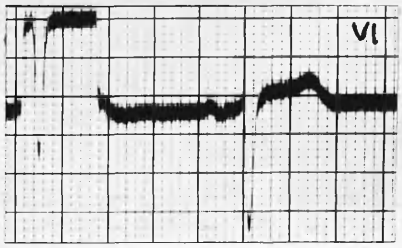


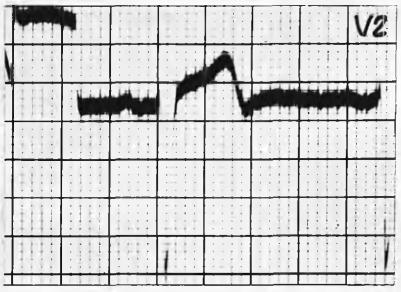


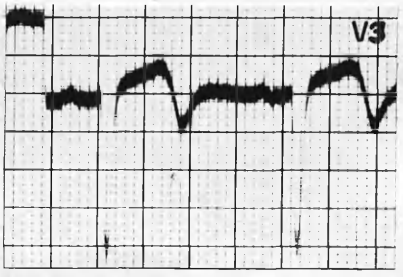
CASE 17, H.H., MALE, 48 YEARS.

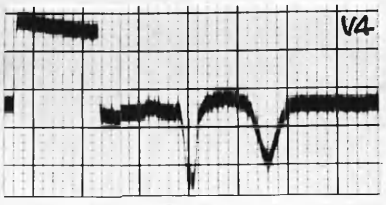
9 WEEKS

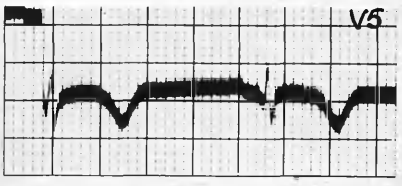
AFTER ONSET.

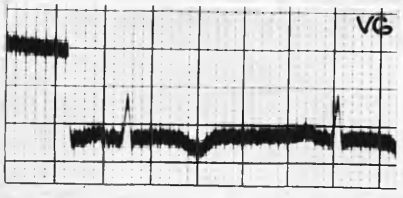


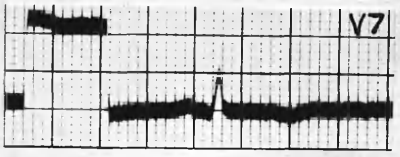












9 WEEKS

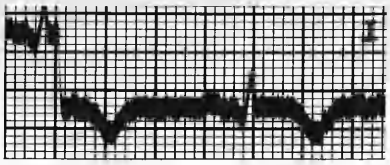
AFTER ONSET.

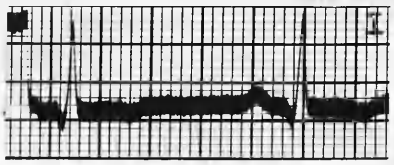
Case 18, A.C., male, 66 years.

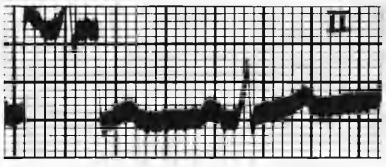
This patient was admitted to hospital one week after the onset of severe substernal pain radiating widely over the chest and back. During the previous month he had had some substernal pain on exertion. Patient was severely ill for the first few weeks of his hospital stay. The chest pain recurred on several occasions. Blood pressure was high on admission, viz. 210/124, but within twenty-four hours it fell to 136/90, and on the following day to 104/74, about which level it remained. The apex-beat was in its normal situation and the cardiac sounds were of average intensity and pure. The chest was of deep bulky shape. Leucocytosis was present. Radiologically the left ventricle and aorta were unduly prominent.

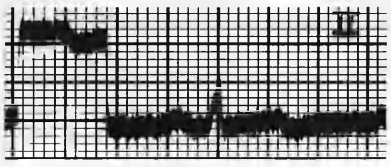
The first set of electrocardiograms shows the typical signs of an acute anterior infarction in lead I, clearly derived from lead VL. The praecordial leads show a deep QS pattern in leads V1 to V4 inclusive and a marginal pattern in lead V5. There is severe elevation of the ST segment in leads V3, V4 and V5. Two weeks after onset the standard limb leads show the usual ST-T evolution. The praecordial leads show corresponding inversion of the T waves in leads V3 to V6 inclusive. The ST-T segments of leads V1 and V2 have now returned to normal but the R wave is still absent,

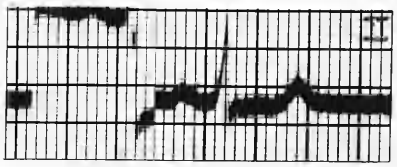
being represented by a slight thickening of the downward limb of the QS deflection. One year and four months after onset the standard limb leads show left axial deviation and shallow inverted T waves in lead I which might be taken to indicate early left ventricular hypertrophy. Lead VL shows typical signs of a local conduction defect of the lateral wall. The praecordial leads show persistent absence of the R wave from V1 to V4 inclusive and slight inversion of the T wave of the apical and axillary leads where, however, there has been increase in the size of the R wave. The late signs in these leads would suggest some degree of left ventricular hypertrophy, either new or unmasked by the regression of the signs of infarction.

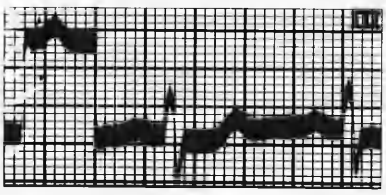


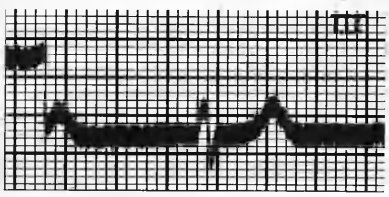


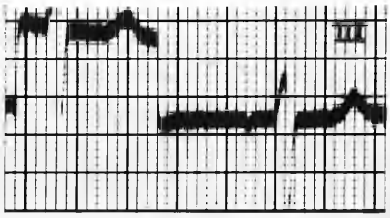


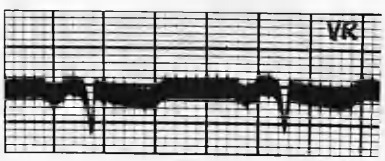


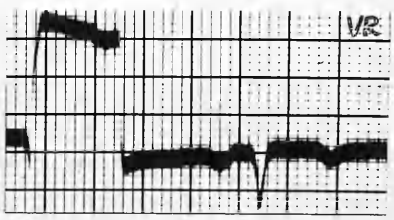


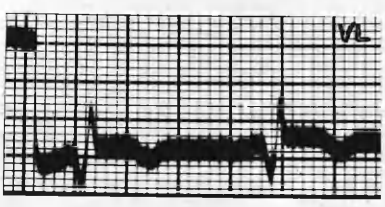


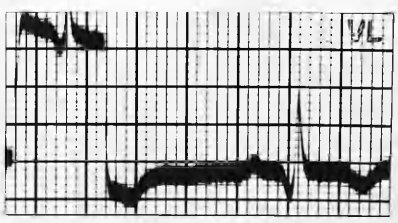


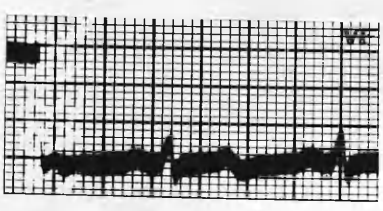


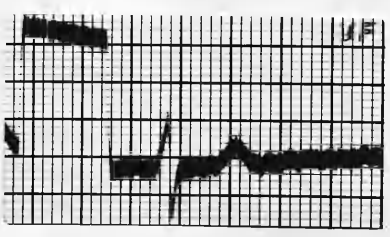










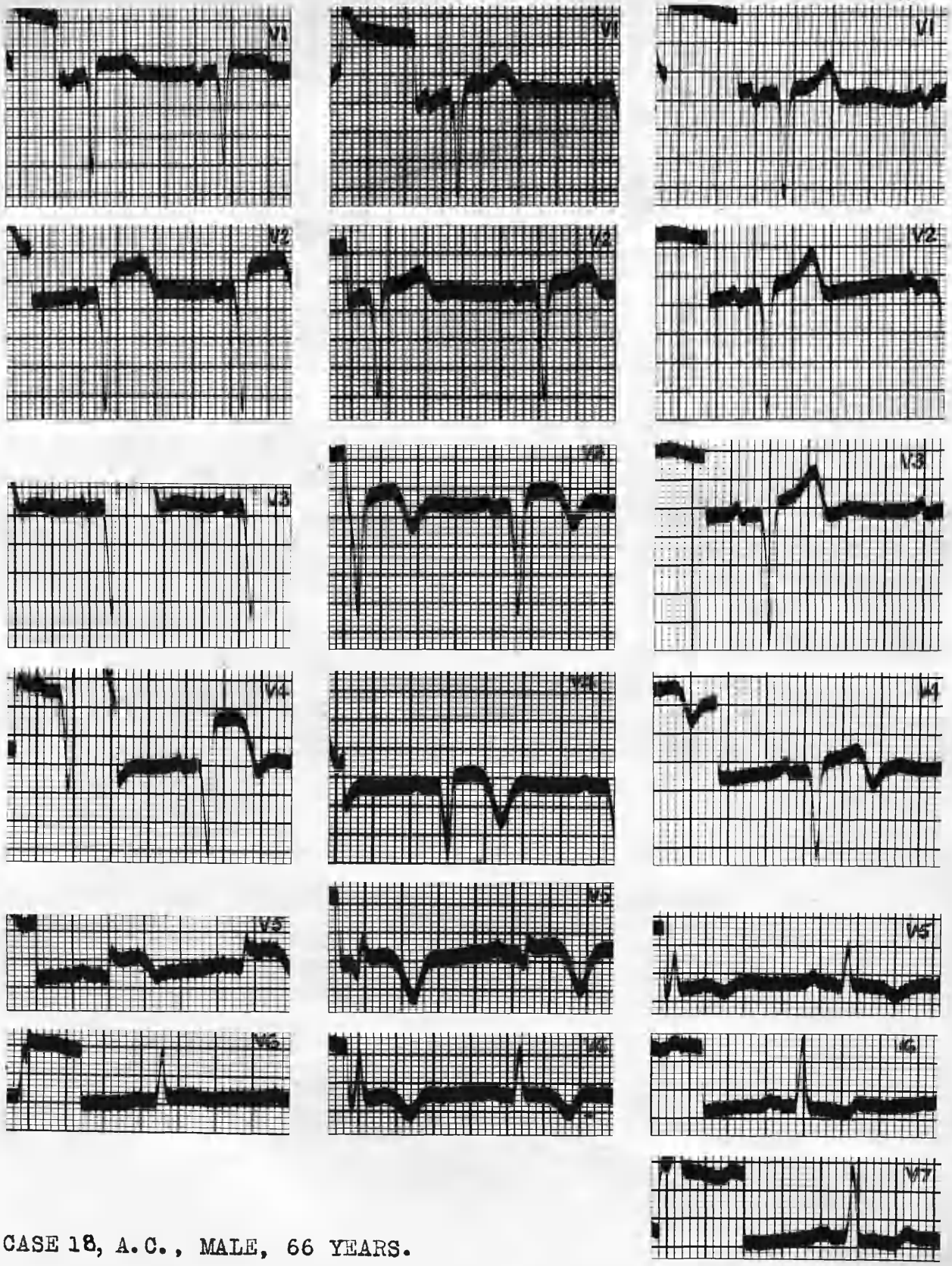


CASE 18, A.C., MALE, 66 YEARS.

1 WEEK AFTER ONSET.

2 WEEKS AFTER ONSET.

1 YEAR 4 MONTHS AFTER ONSET.



CASE 18, A.C., MALE, 66 YEARS.

1 WEEK AFTER ONSET.

2 WEEKS AFTER ONSET.

1 YEAR 4 MONTHS AFTER ONSET.

Case 19, W.L., male, 41 years.

This patient had a typical attack of cardiac pain on his way to work five weeks before his attendance at the out-patient department. Blood pressure was 124/78 and the cardiac sounds were somewhat soft. During the year and a half during which he was kept under observation, blood pressure showed little variation from the above figures. In the later months the apex beat became more difficult to localise and clinically and radiologically there was some increase of cardiac dullness to the left.

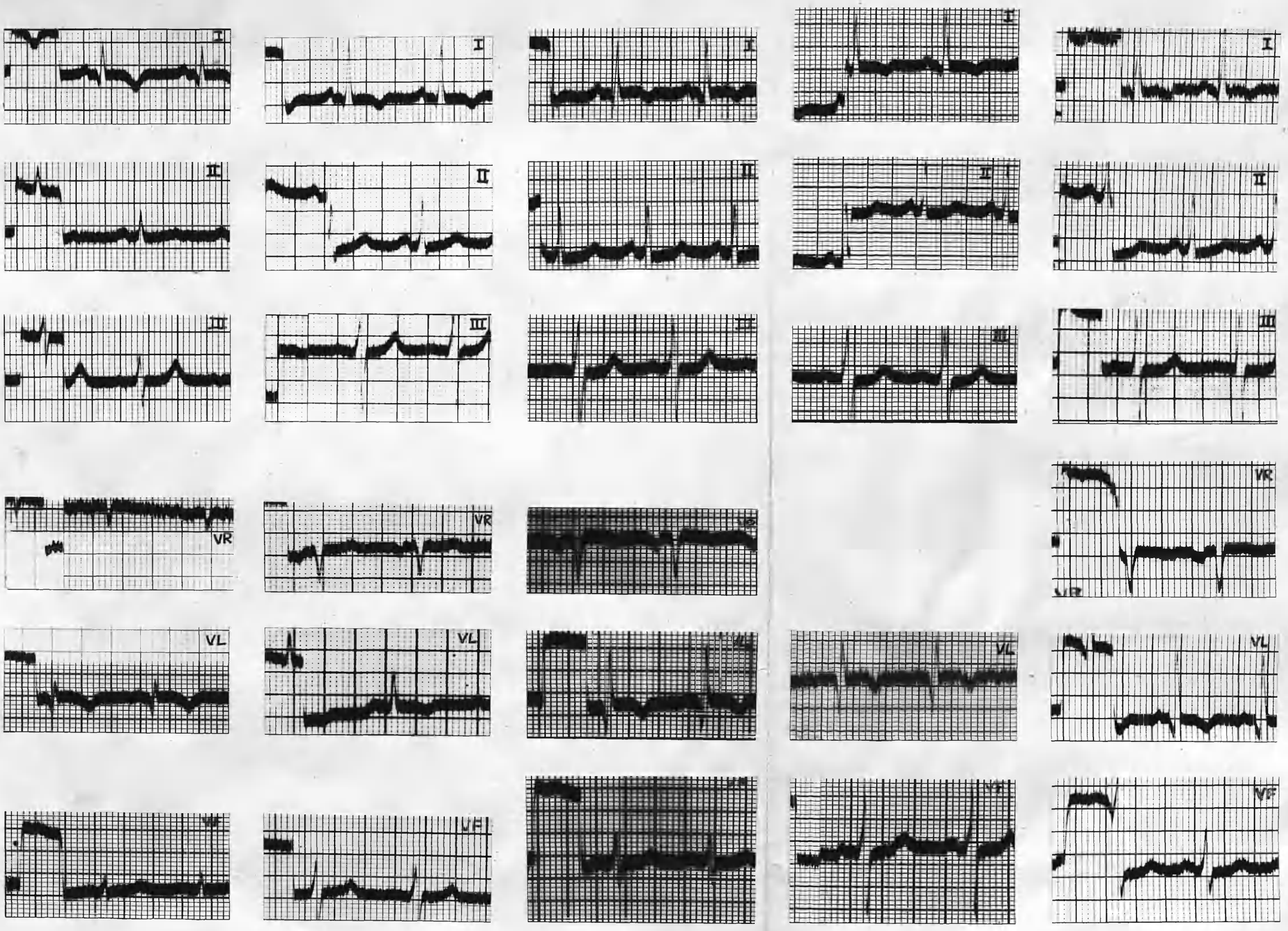
The first electrocardiogram shows in the praecordial leads the typical signs of an anterolateral infarction. The preservation of the small initial R wave in leads V1 and V2 indicates that the septum is intact. The decrease in height of this small R wave from one or two millimetres to nil as the electrode moves from the V2 to the V4 position is diagnostic of anterior infarction. Organisation is proceeding as indicated by the inversion of the T waves in the praecordial leads and in lead VL. Standard limb lead I is typical of an organising anterior infarction.

The next electrocardiogram taken four months after onset shows changes chiefly in leads VL and VF. In so far as the Q wave of lead VL has disappeared, there has been some recovery of function in the subendocardial muscle

subtended by the electrode in the VL position - a recovery also reflected in the increased S wave of lead VF.

Although only a month elapsed before the next electrocardiogram there are further significant changes best assessed from lead VL. The prominent Q wave has returned but not at the expense of the R wave which is taller than in the previous tracing. There is also delay in attaining the peak of the R wave; the ST segment is a plateau and the T wave is sharply inverted. These changes indicate a local conduction defect of the lateral ventricular wall. They are reflected in lead I which is closely similar to lead VL. Evidence of healing is provided by the development of a late R wave in lead V4 which probably represents renewed function in subepicardial muscle.

There is an interval of seven months before the next electrocardiogram but there is little significant change. The final picture, one and a half years after onset, is of left ventricular hypertrophy. The late R wave has increased considerably in lead V4 and has also appeared in lead V3 indicating further regeneration of subepicardial muscle. The signs of local conduction defect in the lateral ventricular wall persist in lead VL.



CASE 19, W.L., MALE, 41 YEARS.

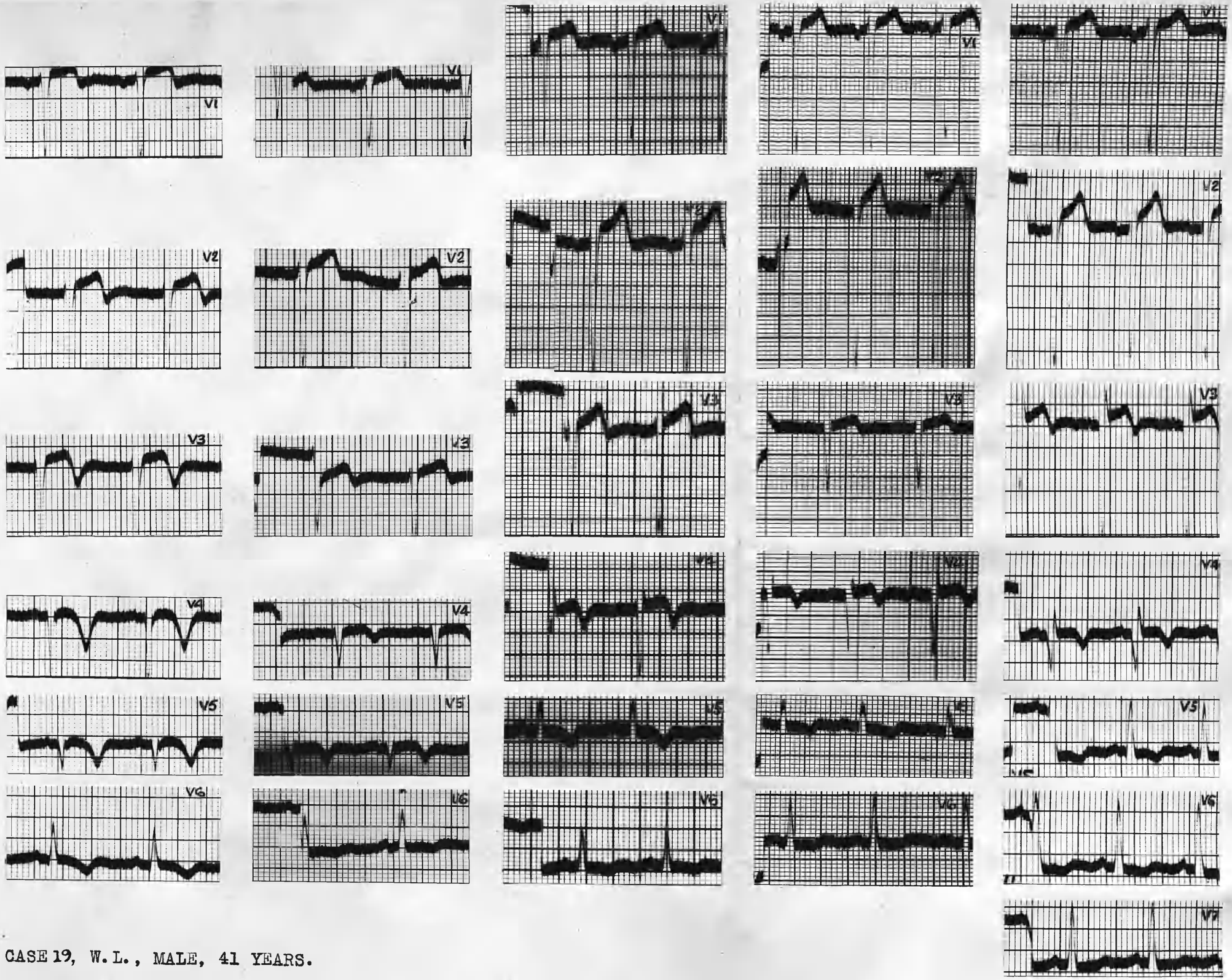
5 WEEKS AFTER ONSET.

4 MONTHS AFTER ONSET.

5 MONTHS AFTER ONSET.

1 YEAR 1 MONTH AFTER ONSET.

1 YEAR 6 MONTHS AFTER ONSET.



CASE 19, W. L., MALE, 41 YEARS.

5 WEEKS AFTER ONSET.

4 MONTHS AFTER ONSET.

5 MONTHS AFTER ONSET.

1 YEAR 1 MONTH AFTER ONSET.

1 YEAR 6 MONTHS AFTER ONSET.

Case 20, A.M., male, 62 years.

This patient first attended the out-patient department eight weeks after the onset of illness. He had been confined to bed at home for most of this period on account of severe substernal pain originally occurring while at work. When seen at the out-patient department patient had been free of chest pain and dyspnoea for a week or so. B.P. was 140/80. The cardiac sounds were pure and the chest was clear. An electrocardiogram at this date shows left axial deviation with flat T wave in lead I as the sole abnormality suggesting the possibility of an anterior coronary lesion. The Q wave although 1.5 mm. deep is only about one fifth of the size of the succeeding R wave and is therefore of equivocal significance. Lead VL shows a borderline Q wave, a slightly delayed R wave and flat T wave probably indicative of local conduction defect. Lead VR has a pattern derived from the back of the heart. Lead VF with its RS form indicates an intermediate to horizontal cardiac position.

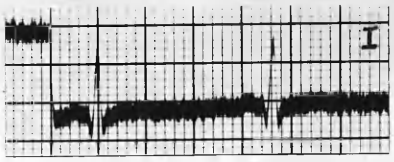
The praecordial leads are typical of an organising anterolateral infarction. The tiny R wave of lead V1 indicates that the septum is intact. Leads V2, V3 and V4 show a central zonal pattern, although islets of active muscle no doubt persist and account for the notching of the QS

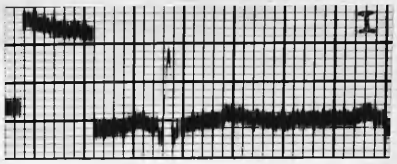
deflection in leads V3 and V4. Lead V5 shows a marginal zonal pattern and in lead V6 there is still upward convexity of the ST segment and shallow inversion of the T wave although there are no longer significant QRS abnormalities. This case illustrates how scanty diagnostic signs may be in the standard limb leads and even in the unipolar leads in the presence of a fairly recent anterolateral infarction as demonstrated by the praecordial leads. The probable cause is the horizontal or semi-horizontal position with counterclockwise rotation such that lead VL on the one hand is not in relationship either to the central or to the marginal zone of infarction and lead VR on the other shows the normal configuration derived from the back of the heart, unaffected by the infarction.

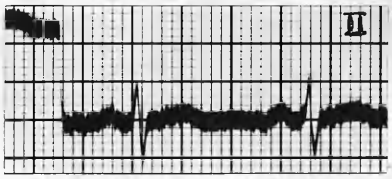
The second electrocardiogram, obtained one year and nine months after onset of illness, shows evolutionary changes in the T waves but the QRS complexes are unaltered. In leads over the right side of the praecordium the T waves have practically returned to normal. In leads V6 (and V7) the ST-T segment is now flat but in leads V4 and V5 the T waves are still sharply inverted. The fixity of the QRS pattern would indicate that any reparative processes in muscle had been scanty.

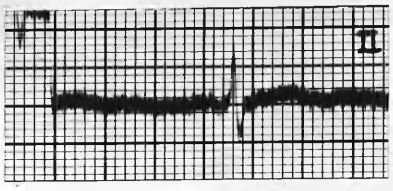
The standard and unipolar limb leads also show some

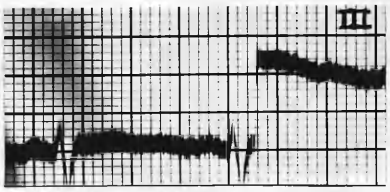
regression of the T wave. In lead VL it is small and upright whence it is transmitted with little change to standard limb lead I. The standard limb leads considered alone give no indication whatsoever of the previous anterior infarction.

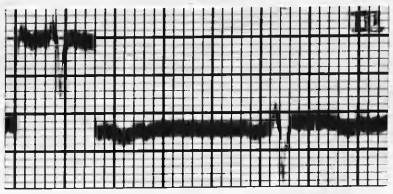


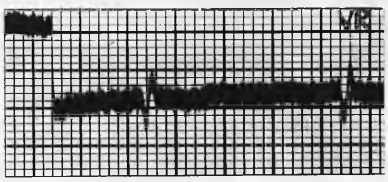


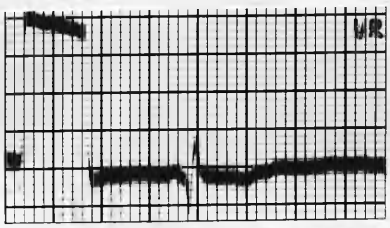


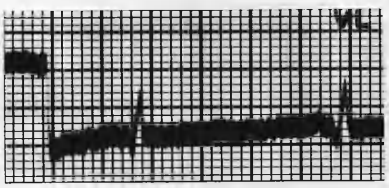


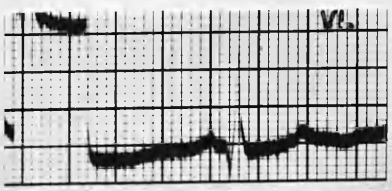


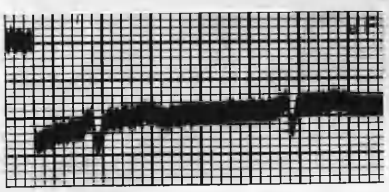


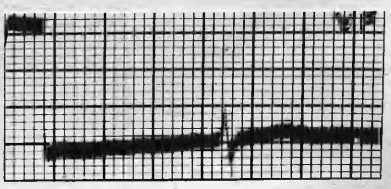








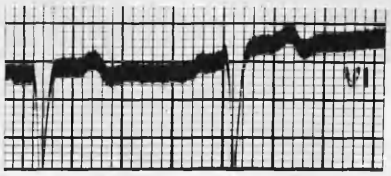


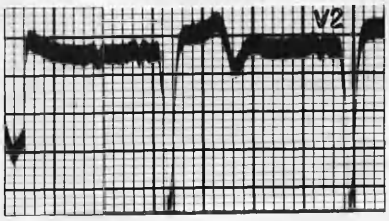


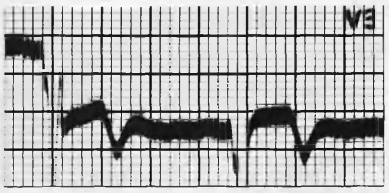
CASE 20, A.M., MALE, 62 YEARS.

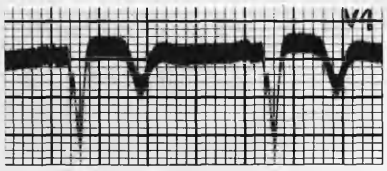
8 WEEKS
AFTER ONSET.

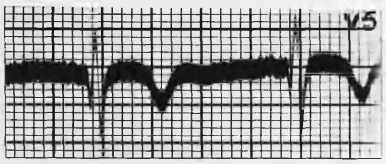
1 YEAR 9 MONTHS
AFTER ONSET.

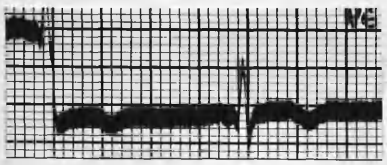








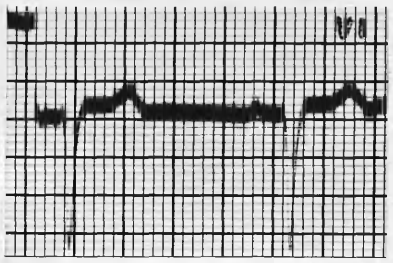


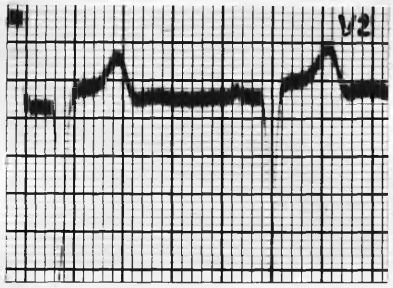


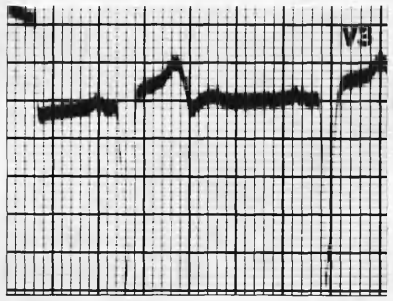
CASE 20, A.M., MALE, 62 YEARS.

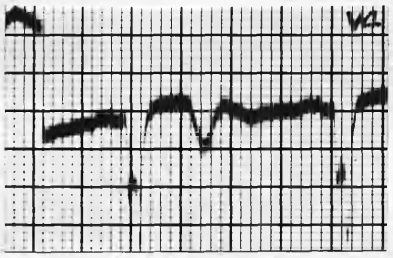
8 WEEKS

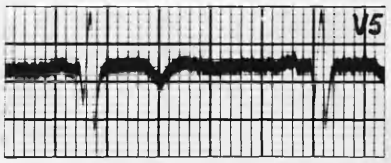
AFTER ONSET.

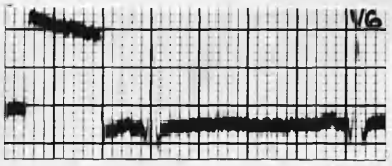












1 YEAR 9 MONTHS

AFTER ONSET.

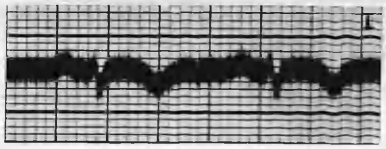
Case 21, R.I., male, 42 years.

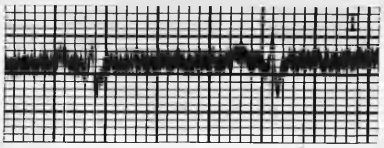
This patient attended at the Out-Patient department eight weeks after an attack of severe praecordial pain with collapse and dyspnoea. The pain lasted for a day. The patient had remained in bed at home for six weeks. Clinical examination was largely negative. The heart sounds were of good quality and blood pressure 150/100. The first electrocardiogram shows signs in the standard limb leads very suggestive of a resolving anterior infarction. The praecordial leads show absence of the R wave in leads V1 to V5, inclusive, and sharp inversion of the T wave in leads V3 to V6, inclusive, justifying the diagnosis of a healting anterolateral infarction. The question as to whether the absence of the R wave from lead V1 indicates horizontal position of the heart as already suggested by the RS deflection of lead VF and the tall R of lead VL or extension of the infarct into the septum cannot be decided.

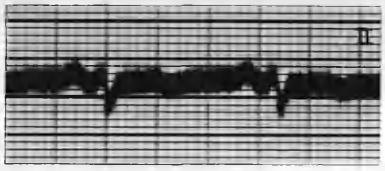
The patient was seen again one year after onset of illness by which time he had returned to work in a factory. The standard limb leads show a satisfactory increase in height of the R wave of lead I, the T wave of which has now become flat. Lead VL shows persistent signs due to infarction which are to a certain extent mitigated

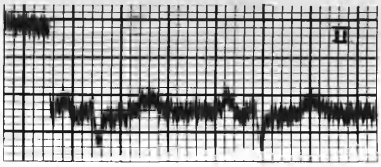
in lead I by the reciprocal of lead VR. The praecordial leads also show partial regression. The ST-T segments of leads V1 and V2 are practically normal and there is some regression in the axillary leads but inversion of the T wave is still conspicuous in lead V4 and to a less degree in leads V3 and V5. The R wave has increased in height in the axillary leads but it has not reappeared in leads V1 or V2. In view of the fact that this heart is probably semihorizontal and that there has been some backward rotation of the apex such that the right arm lead faces the back of the heart, it is probable that the potential variations of the ventricular cavities are referred to the V1 and V2 positions and hence there is no R wave in these leads.

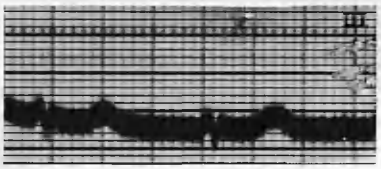
This case shows fairly satisfactory healing although there is clearly scar tissue in the anteroseptal and apical walls.

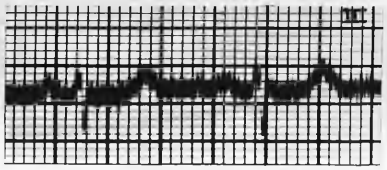


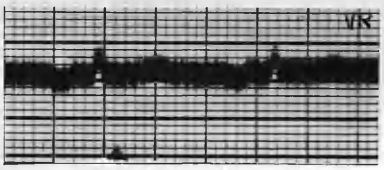


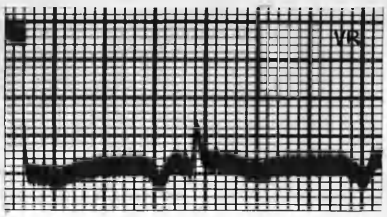


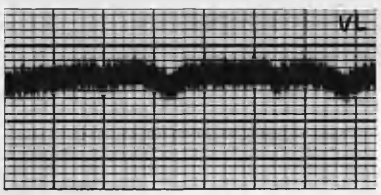


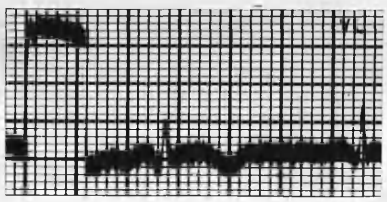


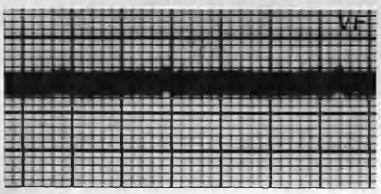


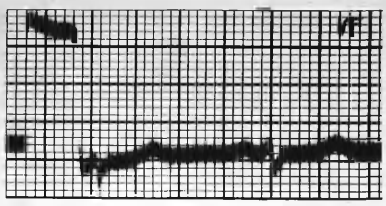








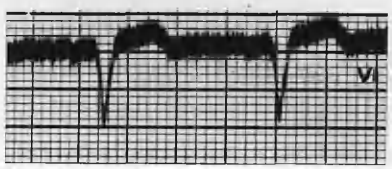


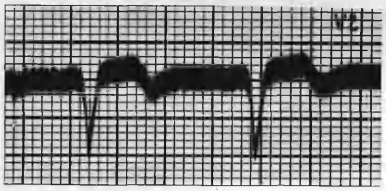


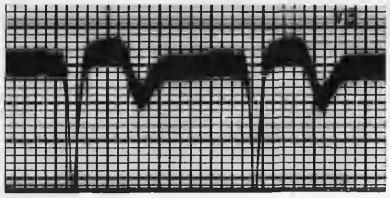
CASE 21, R.I. MALE, 42 YEARS.

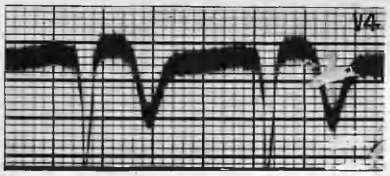
8 WEEKS
AFTER ONSET.

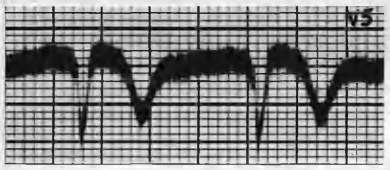
1 YEAR
AFTER ONSET.

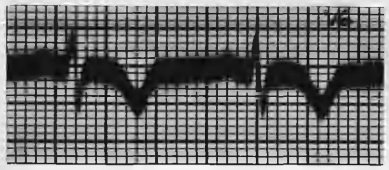


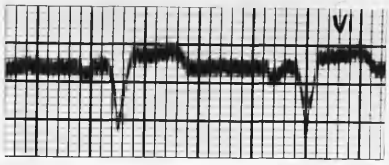


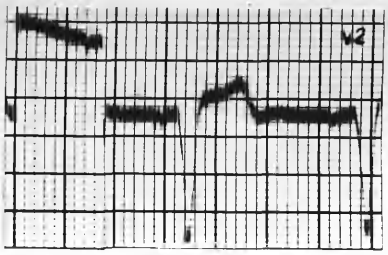


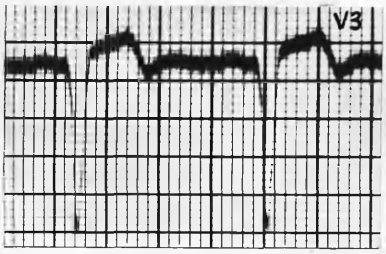


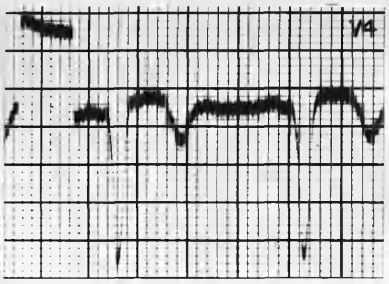


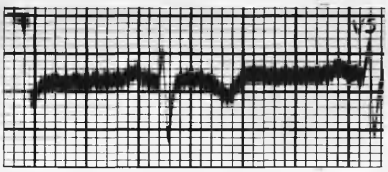


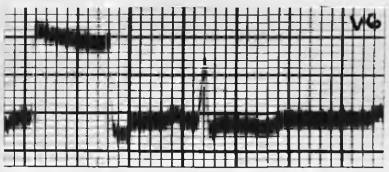


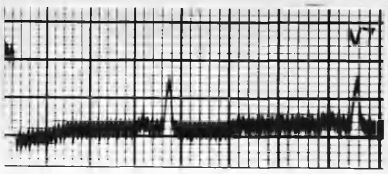












CASE 21, R.I. MALE, 42 YEARS.

8 WEEKS
AFTER ONSET.

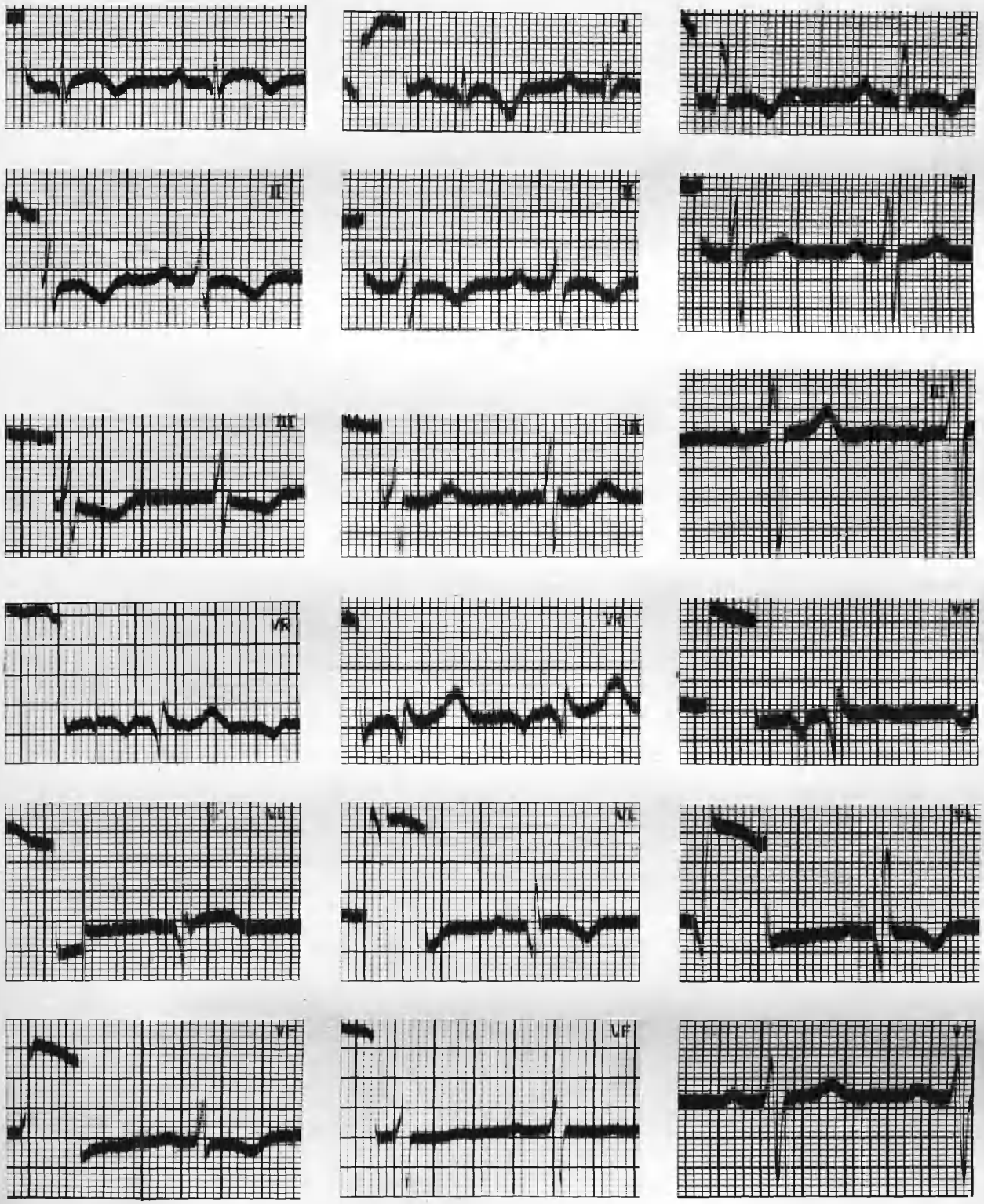
1 YEAR.
AFTER ONSET.

Case 22, I.D., male, 57 years.

This patient, a labourer, complained of tightness in the chest and pain in the praecordium, upper abdomen and left arm which had begun when walking home from work three days before admission to hospital. There were signs of moderate collapse on admission and the cardiac sounds were very soft. Blood pressure fell from an admission level of 150/100 to 115/80. A leucocytosis of 16,000 per c.c. was present. X-ray examination of chest showed some cardiac dilatation and pulmonary congestion.

The standard limb leads show an anterior infarction. Lead VL shows the large Q wave, the small R wave and the elevated ST-T segment typical of involvement of the lateral wall of left ventricle by the infarction. Lead VR which shows a QR pattern clearly derived from the back of the heart also shows a prominent symmetrical upright T wave derived reciprocally from the infarction. Lead VF shows depression of the ST segment which is a typical finding in an unaffected region of the ventricular wall opposite the infarction. The praecordial leads show absence of the R wave in leads V1 to V5 inclusive. The ST-T segment forms a monophasic upright deflection in leads V2, V3 and V4 and is typical of recent infarction of the underlying ventricular wall. From the leads involved, the infarction

is clearly anterolateral with probable extension into the septum in view of the absence of the R wave in lead V1. The evolution of the signs indicates satisfactory progress. By three weeks after onset the R wave of lead VL has increased considerably indicating some local recovery. The final picture six months after onset is that of partial healing. A small R wave has returned to lead V1 indicating septal healing. Leads V3 and V4 remain frankly abnormal in so far as there is no R wave and in the axillary leads the R wave remains abnormally small. Lead VL shows a conduction defect of the lateral wall of the left ventricle. At this time, i.e. six months after onset of illness, the patient still felt tightness across the front of the chest on exertion. There was slight cyanosis. The apex beat was faintly palpable and the cardiac sounds were of medium intensity.

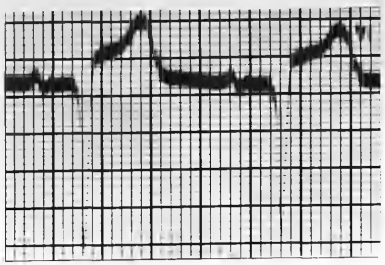


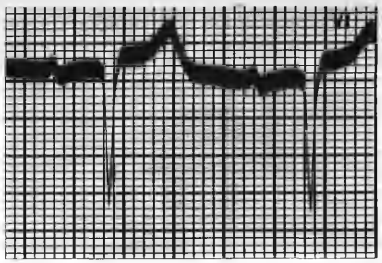
CASE 22, I.D. MALE, 57 YEARS

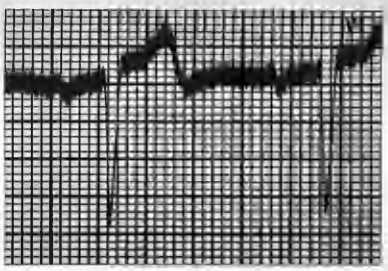
12 DAYS
AFTER ONSET.

3 WEEKS
AFTER ONSET.

6 MONTHS
AFTER ONSET.


V


V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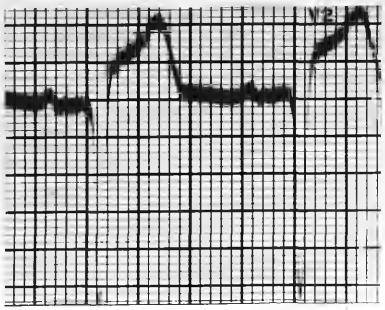
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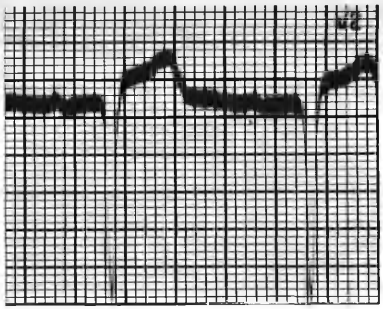
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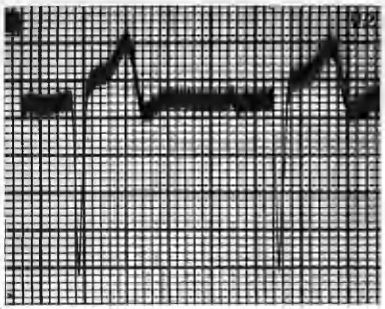
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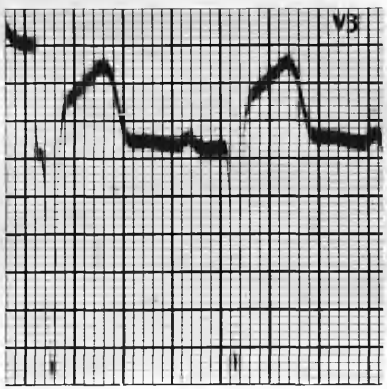
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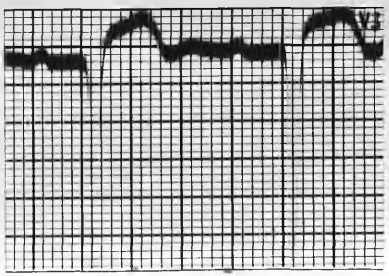
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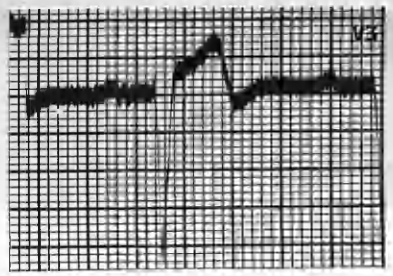
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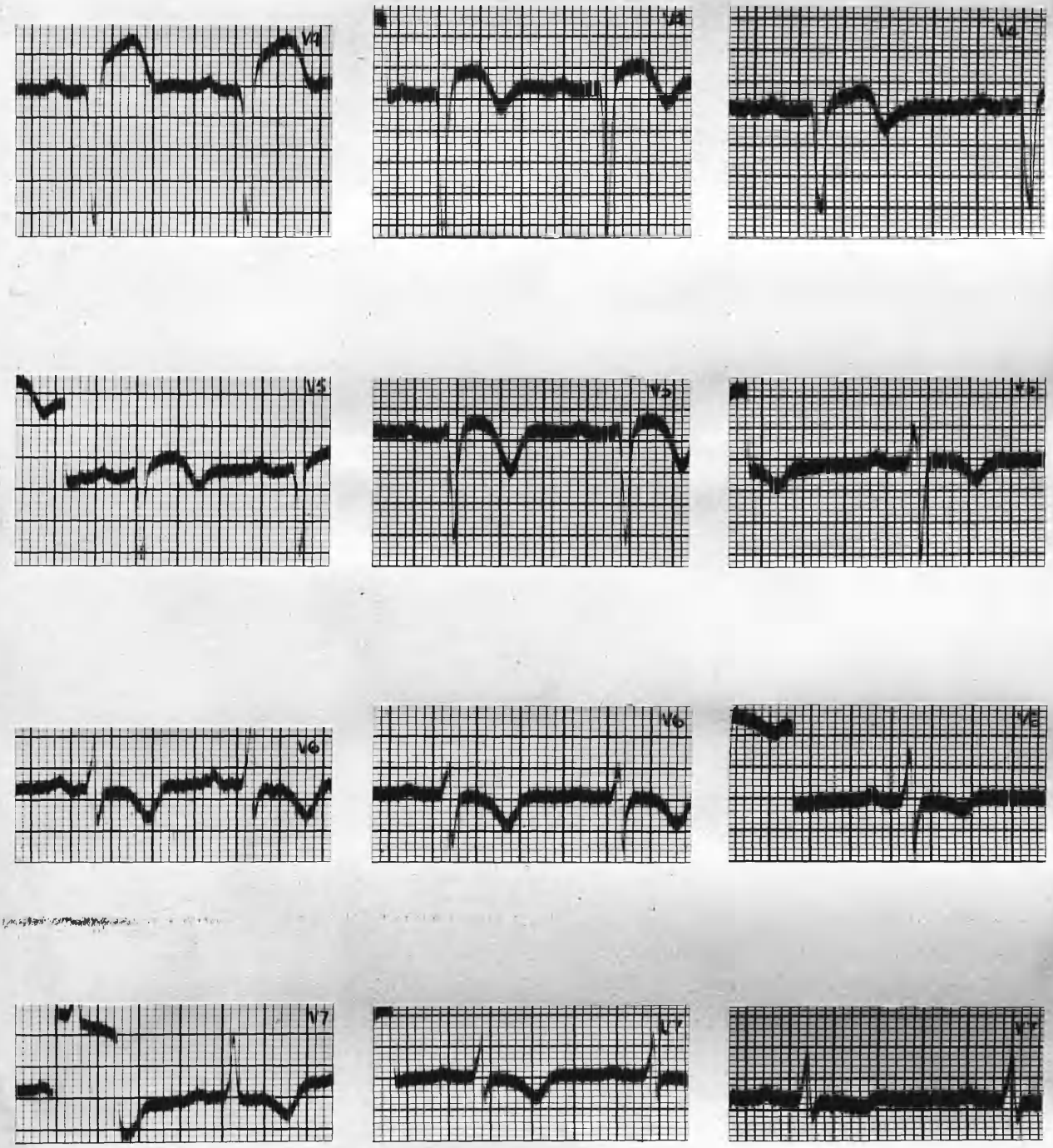


CASE 22, I.D. MALE, 57 YEARS

12 DAYS AFTER ONSET.

3 WEEKS AFTER ONSET.

6 MONTHS AFTER ONSET.

Case 23, J.McA. male, 64 years.

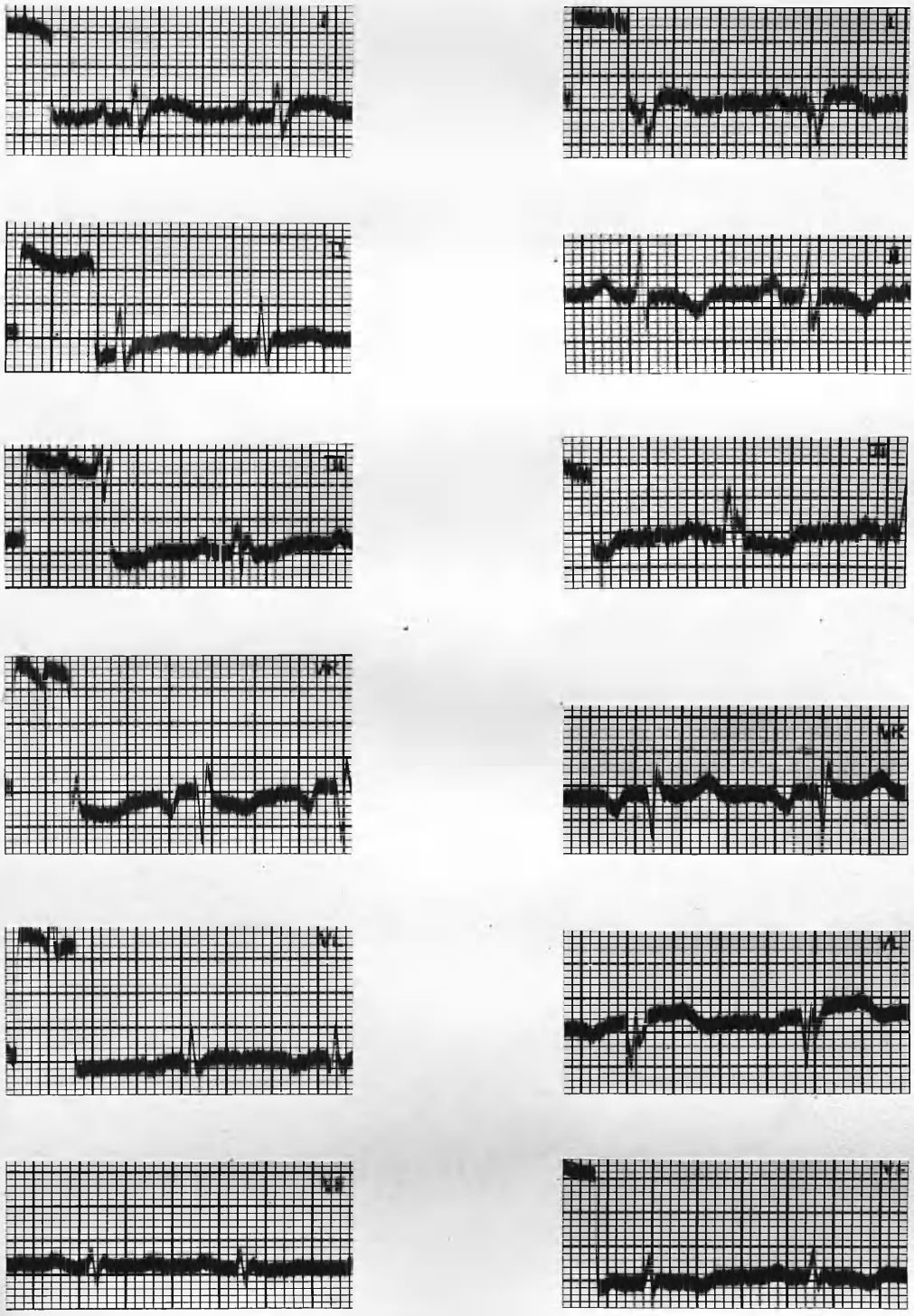
This patient had a severe attack of cardiac pain with collapse on the day before admission to hospital. Pericardial friction was audible on the first two days of his hospital stay. Blood pressure fell from an admission level of 152/98 to 100/62 two days later. Cardiac sounds were invariably of poor quality, chest pain recurred for several days and cyanosis and orthopnoea persisted for a few weeks. The chest showed signs of bronchitis. Radiologically the heart shadow was within normal limits. Previous to his present illness the patient had had angina of effort for four years.

The first 13-lead electrocardiogram was obtained four days after onset. The standard limb leads show slight broadening of the QRS deflection especially in lead 3. The ST-T segment is elevated and the T wave is not discernible in lead I. There is no Q wave however, so that the diagnosis of anterior infarction is not conclusive since pericarditis as such cannot be ruled out. Lead VL shows a small Q wave, only 20% however of the succeeding R wave. There is slight elevation of the ST segment but it is clear that the conspicuous elevation of the ST segment in lead I is chiefly due to the reciprocal of its frank depression in lead VR which is however typical of a lead facing the endocardial

surface of an infarct. The ventricular pattern in lead VR suggests that it is derived from the back of the heart. Praecordial leads show an extensive infarction involving the anteroseptal wall and encroaching onto the anterolateral as seen by the marginal QR complex in lead V5. The elevation of the ST segment in this lead is no doubt due to extension of the pericarditis from the central zone of transmural infarction.

As indicated in the clinical notes, this patient was sharply ill during the first few weeks of his hospital stay. The next electrocardiogram taken two weeks after onset shows that infarction has spread laterally. There are now signs of severe infarction in lead VL, the Q wave has become very prominent, the R wave is small and there is conspicuous ST-T elevation. Lead VF being opposite this extension shows slight ST depression as does also standard limb lead III, but lead VR is clearly not influenced by this lateral extension. The disappearance of the ST depression and the symmetrical upright T wave indicate satisfactory evolution of that part of the infarct from which they are derived. The standard limb leads are now diagnostic of anterior infarction since a significant Q has now developed and the R wave has become greatly reduced in size. The praecordial leads show the lateral

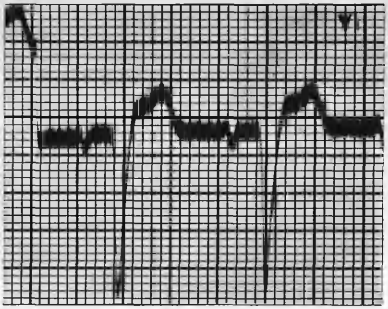
extension at the V5 position where the R wave is now reduced to a notch. The elevation of the ST segment is still conspicuous in leads V5 and V4 but it has decreased in the anterior praecordial leads. This indicates some progress in healing in this situation although there is no return of the R wave. This case shows spread of infarction in the anterolateral wall in thickness and probably also in extent while there are signs of healing in the more anterior parts of the infarction. In view of the fact that the unipolar limb leads derive their signs from both regions it would be difficult to evaluate the standard limb leads without the help of the unipolar leads.

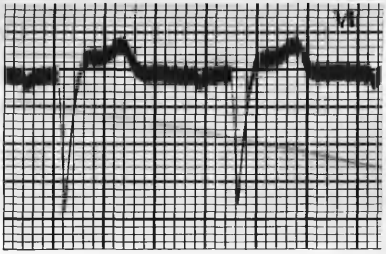


CASE 23, J.McA. MALE, 64 YEARS.

4 DAYS
AFTER ONSET.

2 WEEKS
AFTER ONSET.


V1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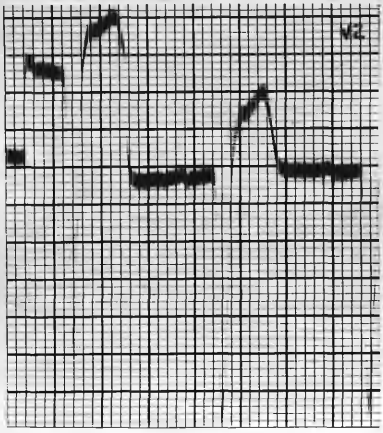
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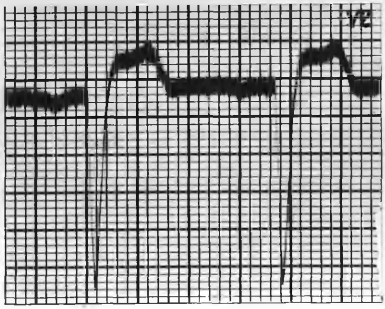
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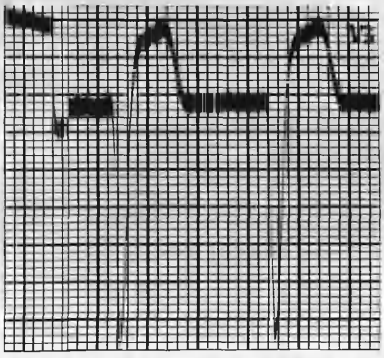
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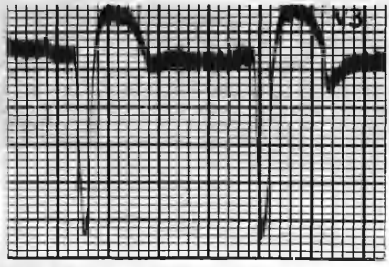
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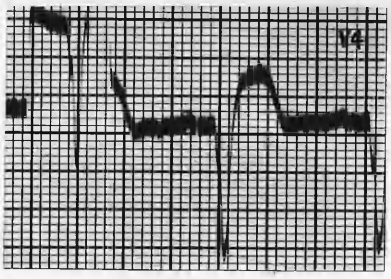
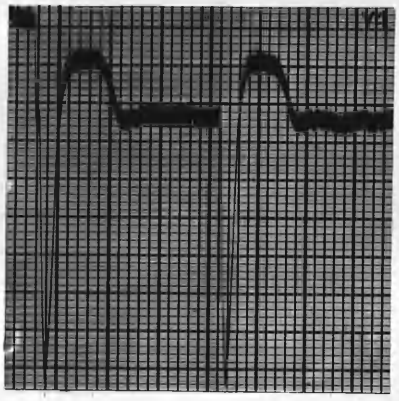
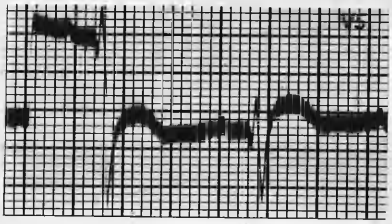
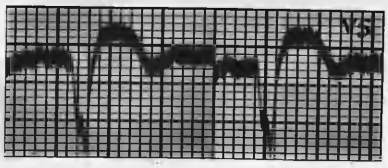
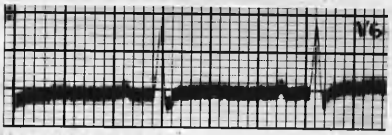
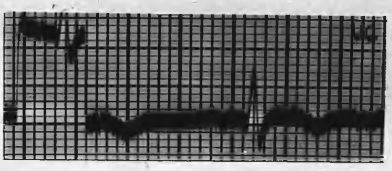


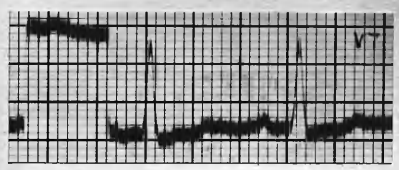


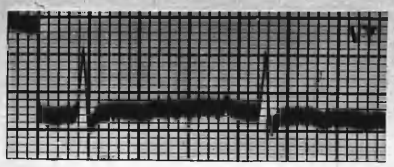












CASE 23, J.McA. MALE, 64 YEARS.

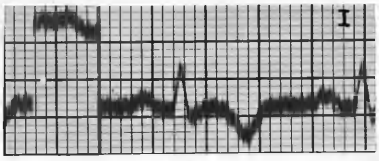
4 DAYS
AFTER ONSET.

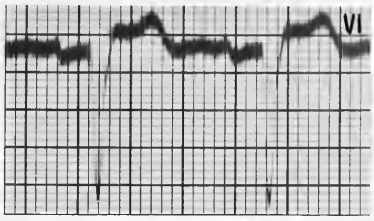
2 WEEKS
AFTER ONSET.

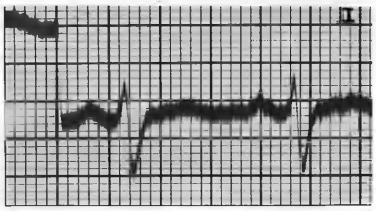
Case 24, G.G., male, 56years.

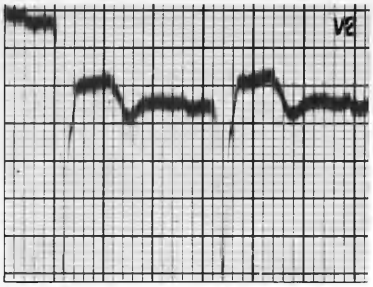
This patient had been ill at home for nine months before he was sent to the out-patient department. He complained of dyspnoea on exertion and praecordial discomfort. Blood pressure was 140/90. There was diffuse praecordial pulsation which was expansile over the third right interspace and radiologically paradoxical pulsation could be demonstrated in this region of the cardiac silhouette. The diagnosis of an aneurysm of the anterolateral wall of the heart was therefore made. The standard limb leads show broadening of the QRS complex of all leads and deep inversion of the T waves of lead I, the picture suggesting early left bundle branch block. The unipolar leads show that the heart is semihorizontal or horizontal to judge from the RS complex of lead VF and the tall R of lead VL. However this lead shows a thick short Q wave and a delayed R wave suggesting a local conduction defect with which the prominent inverted T wave is also consistent. Lead VR faces the back of the heart as seen by its QR deflection and inverted P wave. The praecordial leads show deep QS deflections in leads V1 to V4 inclusive and in a lead taken over the site of expansile pulsation in the third left interspace. The axillary leads show small R waves. The form of the RS deflection in the axillary leads indicates that the /

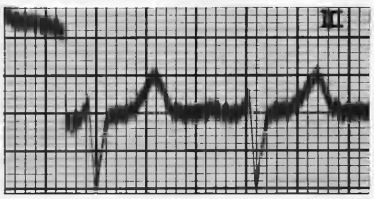
/ the transition zone is well to the left because of backward rotation of the apex. For this reason also the potentials of the ventricular cavities may be referred to the V1 position and account for the absence of the R wave there. The persistence of a QS deflection in V3, V4 and probably also in V2 is due to absence of muscular tissue in the subjacent ventricular wall. This case clearly shows how a poorly healed anterolateral infarction is associated with a definite cardiac position, viz. horizontal or semihorizontal with backward rotation of the apex so that the transition zone is at the V5 or even V6 position and so that lead VR faces the back of the heart. It is probable that the imbalance produced by the impaired contractility of the anterolateral wall along with the unaffected contractile power of the posterior wall is a powerful agent in establishing this cardiac position.

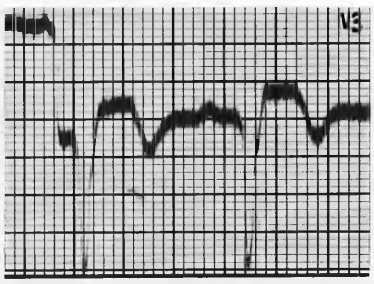


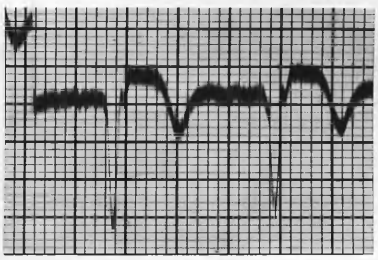




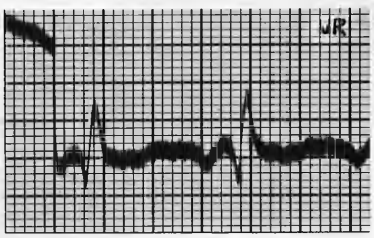


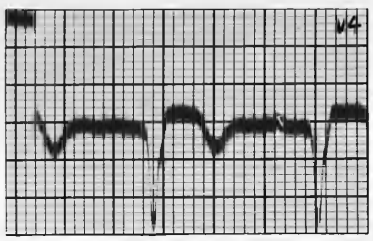


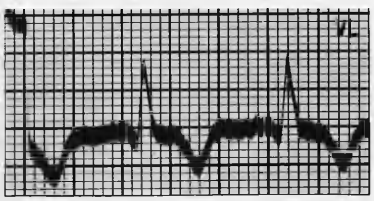


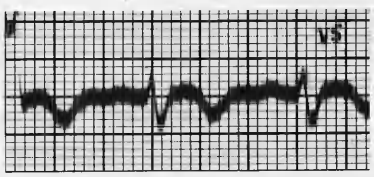


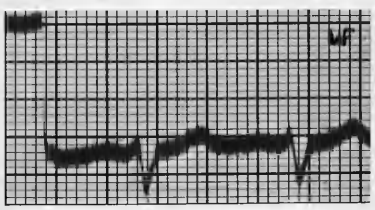
OVER 3rd LEFT INTERSPACE.

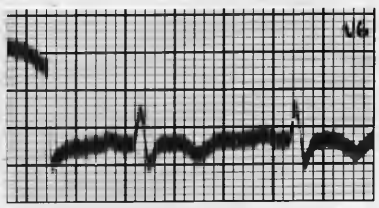


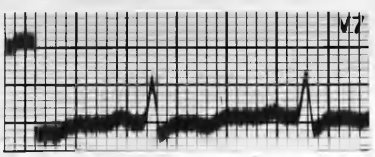












CASE 24, G.G. MALE, 56 YEARS.

9 MONTHS AFTER ONSET.

Case 25, R.W., male, 44 years.

This patient was awakened in the early hours of the first day of illness by the sensation of "iron bands" across his chest. He had also pain in his left arm. Morphine was given with good effect. He was removed to hospital on the third day of illness, the praecordial pain persisting in modified degree. He recalled that a year previously he had had constriction in the chest with pain down the left arm, lasting for a week.

On admission he showed greyish pallor but other signs of shock had disappeared. B.P. was 130/90 about which level it remained during his hospital stay. The cardiac sounds were soft and pure. The patient made a very satisfactory recovery and was able to resume work later as a civil engineer.

The first electrocardiogram taken three days after onset shows as its sole abnormality moderate elevation of the ST segment in lead I; the T wave is still upright. The QRS complex of lead I is very small. No specific abnormality is seen in the unipolar limb leads which are typical of a vertical heart. The praecordial leads show absence of the R wave from lead V1 to V5 inclusive. The prominent diphasic contour of the P waves of leads V1 and V2 suggests that the electrode was in the vicinity of the right auricle

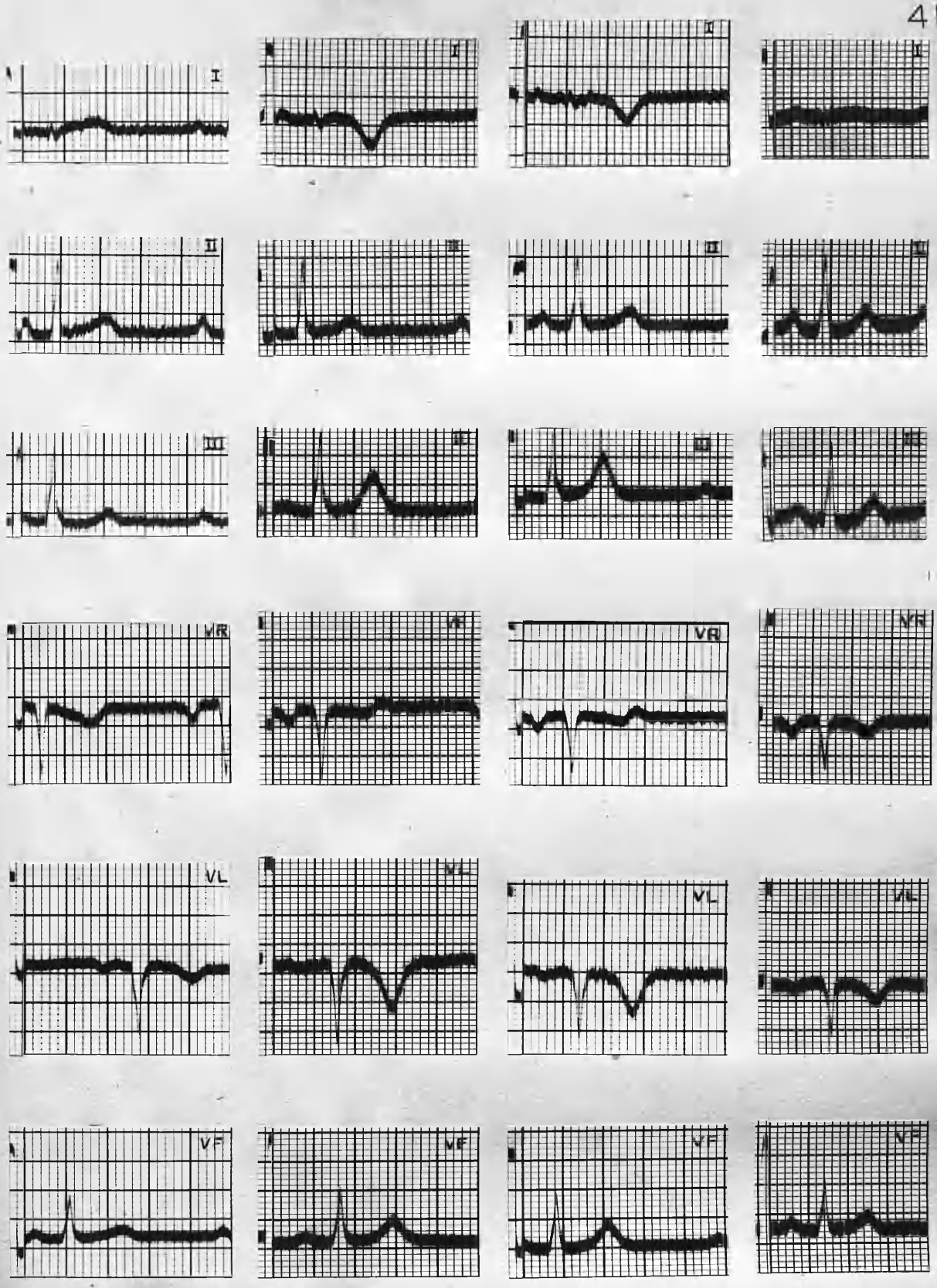
and hence that the ventricular complex of these leads is derived chiefly from the right side of the septum and the right ventricular wall and not from the ventricular cavities; hence their absent R waves and long QS deflections probably indicate involvement of the septum. Similar deflections are seen in leads V3 and V4 but in lead V5 there is a sudden change to a very small splintered QS deflection. The elevation of the ST junction is greatest in lead V3 but there is already inversion of the end of the T wave in this lead. Although the early phase of the ventricular complex is represented in lead V6 solely by a small R wave, slight elevation of the ST segment is still detected as far to the left as this lead is. These leads indicate an extensive infarction involving the septum and anterolateral wall of the heart.

During the next eleven days the evolution of the T wave is progressing simultaneously with return to the isoelectric level of the ST segment. Lead VL shows a deep cove plane T wave which accounts for a similar T wave in standard limb lead I. Very deep T waves reaching 16 to 17 millimetres below the isoelectric level are seen in leads V3 and V4. Even as early as two weeks after onset the R wave is regaining height in leads V6 and V7 and there is shortening of the QS deflection in leads V3 and V4.

In electrocardiograms obtained three and a half

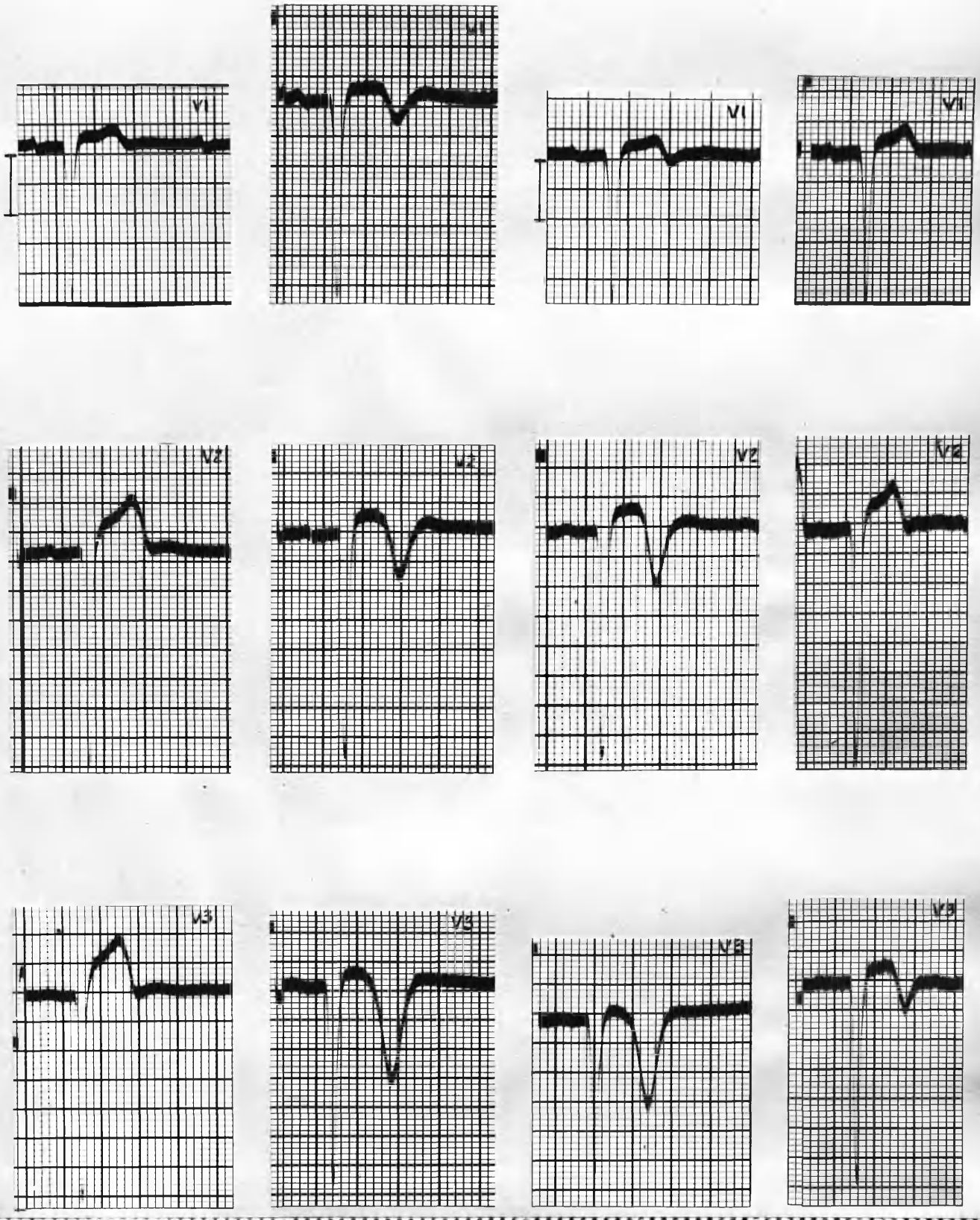
weeks after onset, regression of the T waves is evident. They are less negative in lead V1 over the right praecordium and in the apical and axillary leads; in lead V7 and T wave is positive again. There is little difference however in the T waves of leads V2 and V3.

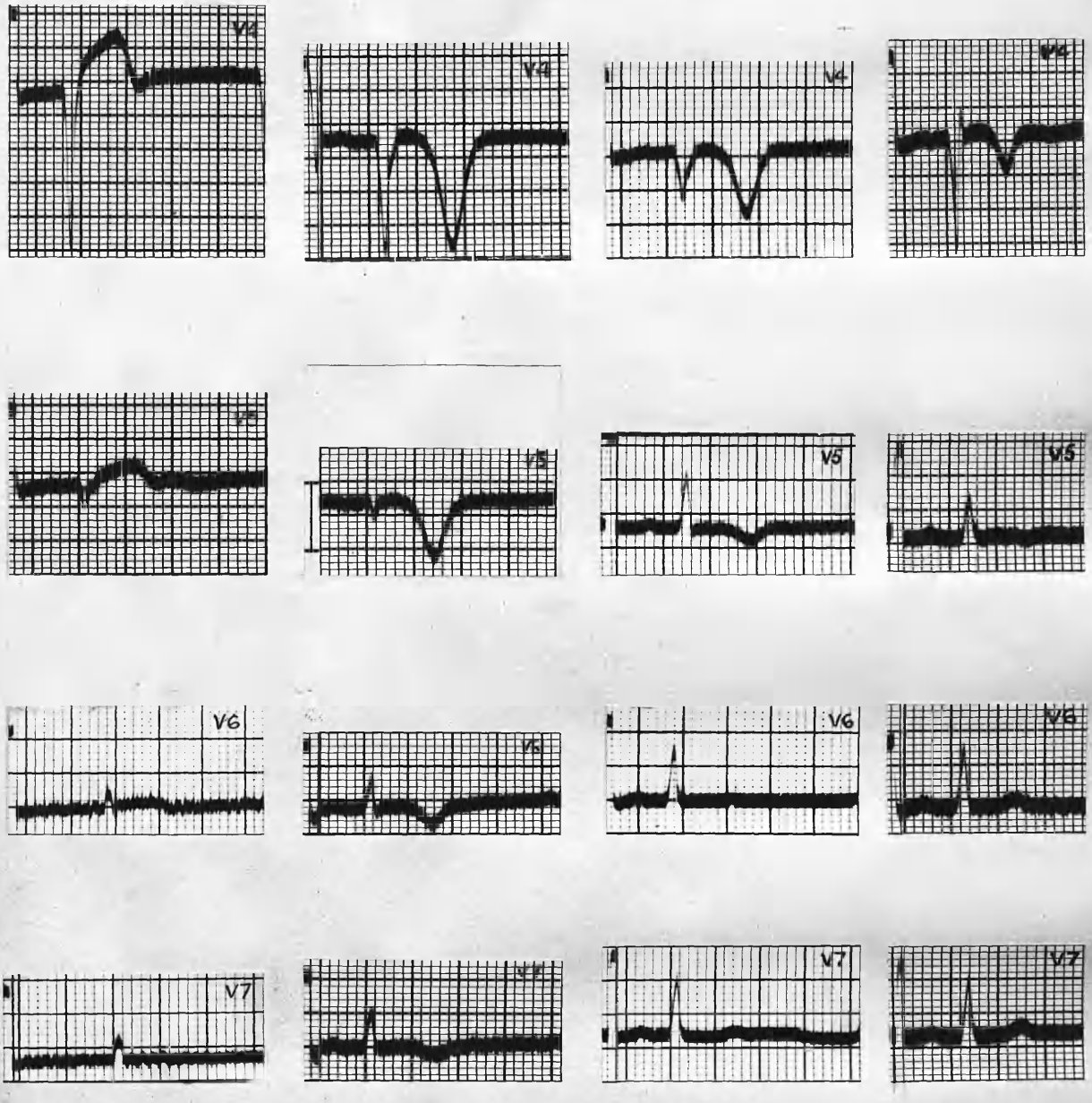
At twenty-eight weeks after onset, the standard limb leads are still abnormal in so far as the QRS complex of lead I is very small and the T wave, although positive, is low. The unipolar limb leads are normal for a vertical heart. The initial R wave of the praecordial leads remains absent but in lead V4 a late R wave has developed, measuring 3 millimitres, preceded by a deepened Q wave. The ST segment is now isoelectric again and the T wave remains inverted only in leads V3 and V4. Abnormal signs have practically disappeared from the axillary leads. Healing would appear to be very satisfactory in the leftward margin of the infarction. At the V4 level there is evidence of functional recovery of subepicardial muscle but at the V3 and V2 levels there is probably scarring with little regeneration.



No25, R.W., MALE, 44 YEARS.

3 DAYS AFTER ONSET.	2 WEEKS AFTER ONSET.	3½ WEEKS AFTER ONSET.	28 WEEKS AFTER ONSET.


V1
V1
V1
V1
V2
V2
V2
V2
V3
V3
V3
V3



No 25, R.W., MALE, 44 YEARS.

3 DAYS AFTER ONSET.	2 WEEKS AFTER ONSET.	3½ WEEKS AFTER ONSET.	28 WEEKS AFTER ONSET.

Case 26, W.A., male, 42 years.

This patient, a mine manager, came under observation five months after an attack of myocardial infarction, confirmed electrocardiographically and for which the patient remained under treatment at home.

When he was examined at the out-patient department, he presented no cyanosis or dyspnoea. Blood pressure was 130/80. There was no clinical cardiac enlargement and the sounds were of average intensity and pure. The patient did admit however that he had substernal pain now and again on exertion.

An electrocardiogram at this date shows a significant Q wave in standard limb lead 1 and lead VL combined with shouldering of the ST segments and inversion of the T waves. The praecordial leads show deep QS deflections in leads V2, V3 and V4 and significant Q waves in leads V5 and V6 with inversion of the T waves in all the praecordial leads. These signs indicate an extensive anterolateral infarction.

After a lapse of three months, the clinical condition remained satisfactory. There is some evolutionary change at the V6 level - the Q wave is smaller, the shouldering of the ST segment has disappeared and the T wave is now upright. Interest however centres on lead VF which now shows a QR complex whereas, previously

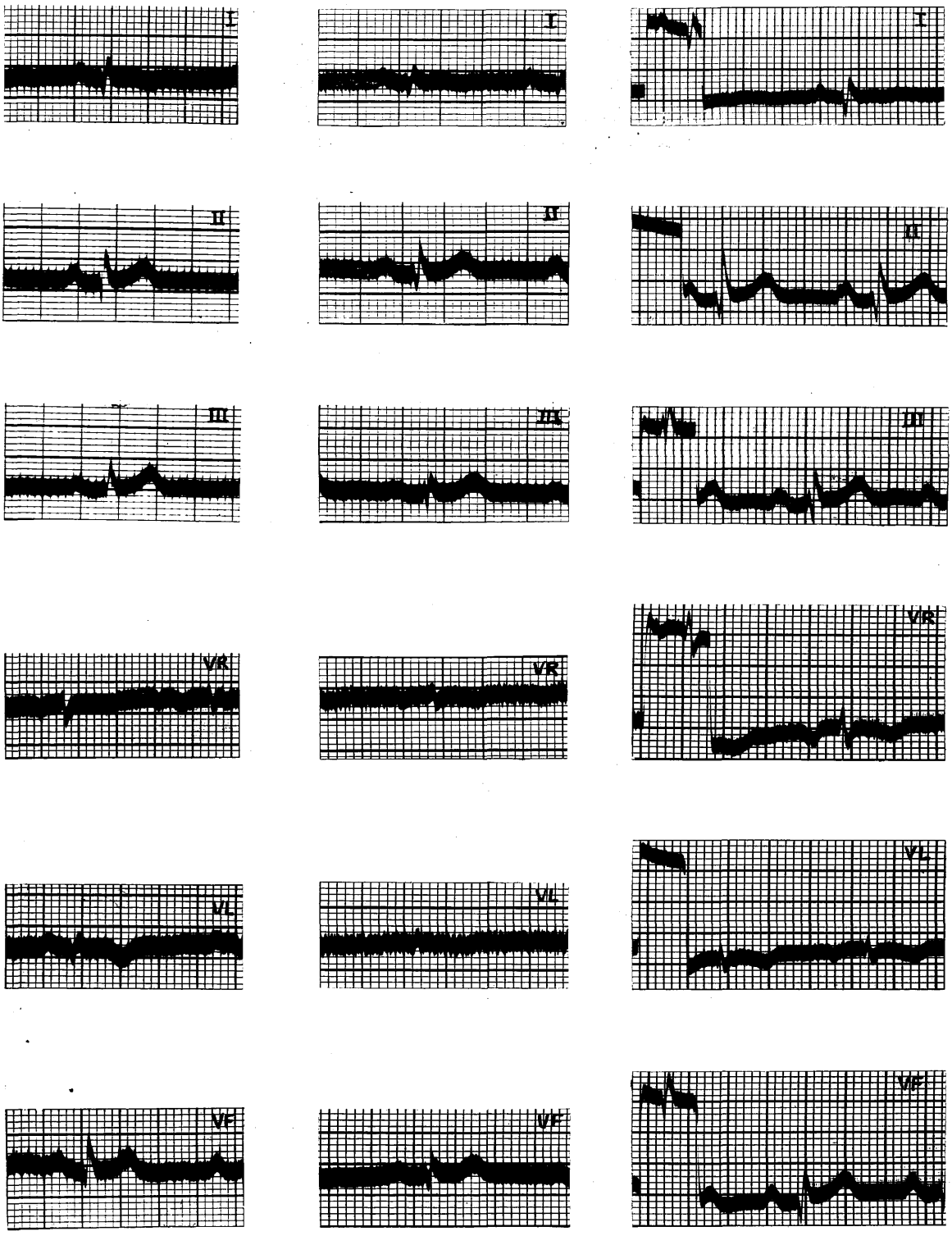
/ its Q wave was minute and its R wave prominent. Concomitantly, lead VR is showing a small RS complex instead of a chiefly negative complex previously.

The position is however clarified by the tracings taken one and a half years after onset by which time patient had resumed business and was but little incommoded by chest discomfort or dyspnoea. As before the praecordial leads show the pattern of a large anterolateral infarction, apparently now fixed. Lead V6 continues to show a marginal QR pattern, with, however, elevation of the RS-T junction which when associated with a concavely upward RS-T segment and upright T wave, as it is in this instance, may be within normal. At this late healed stage, the pattern of lead VF is that derived from an intermediate heart. Its prominent Q wave is fifty per cent of its succeeding R wave; its duration from onset to nadir is only 0.02 sec. The high RS-T take-off, which was not present in tracings taken nearer the acute stage of illness, is probably within normal, especially in view of the shape of the RS-T segment, viz. concave upwards. The T wave is large, upright and symmetrical and may be exaggerated by the reciprocal effects of the large infarction in the anterior wall. Apart from this influence on the T wave, the pattern of lead VF is within normal for an intermediate

heart. Insofar as the Q wave is an early transient deflection and in intermediate hearts is probably the result of septal activation, its reappearance at this stage would suggest healing of a septal extension of the infarction. This interpretation is favoured by the return of the early R wave in lead VR which may have the same genesis. It is noteworthy however that that part of the septum subtended by the electrode at the V1 position was unaffected since a normal r wave has been retained throughout.

Thus the intermediate cardiac position is largely responsible for the patterns in standard limb leads II and III. When lead VR in reciprocal is added to lead VF to form lead II, all the deflections are exaggerated. A very similar additive effect is seen in standard limb lead III except that there is a tiny Q wave in lead VL which supplies the very small, early R wave (as opposed to the later R^1 wave) of lead III. Thus in standard limb leads II and III, the effects of the anterolateral infarction are some exaggeration of the upright T waves and the minute early R wave of lead III. Finally in lead I, the Q wave, more prominent than before, is largely the reciprocal of the normal R wave of lead VR in view of the minuteness of the abnormal Q wave of lead VL. The

chief abnormality in lead I is the smallness of the R wave, for the heart is not vertical which would have accounted for it, otherwise. The flat T wave is abnormal being due to a summation of the abnormal inverted T wave of lead VL with the probably normal inverted T wave, in reciprocal, of lead VR. Thus the stigmata of previous anterolateral infarction in lead I are the small R wave and flat T wave, not the Q wave.

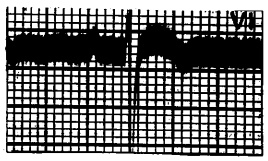


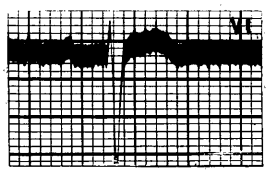
CASE 26, W.A. MALE, 42 YEARS.

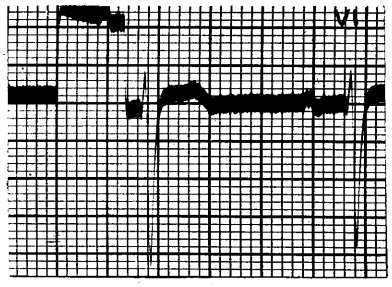
5 MONTHS AFTER ONSET.

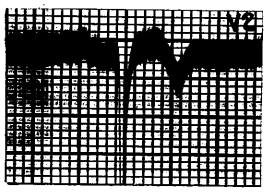
9 MONTHS AFTER ONSET.

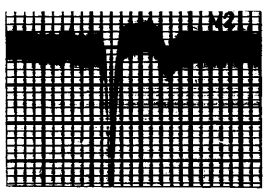
1½ YEARS AFTER ONSET.

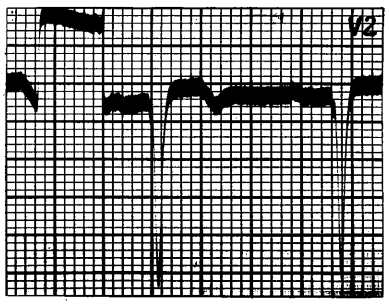


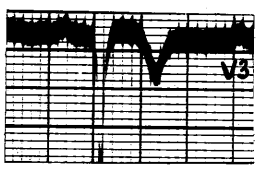


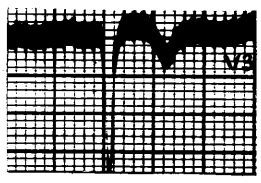


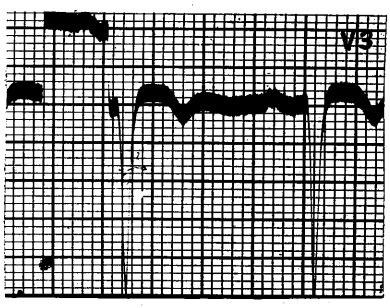


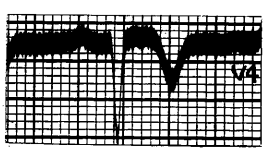


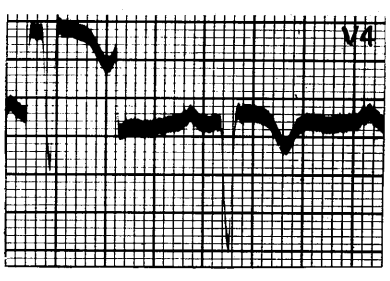


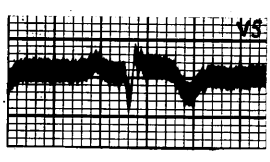


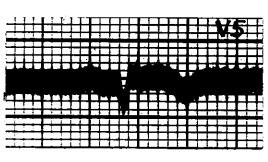


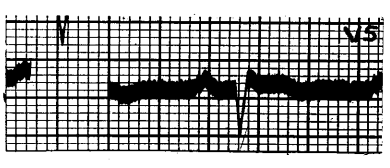


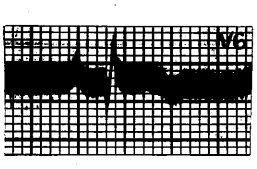


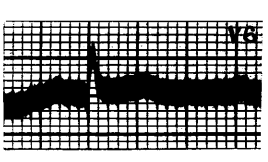


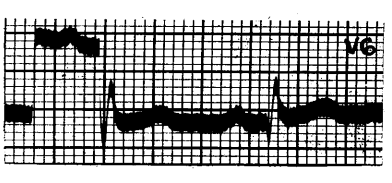


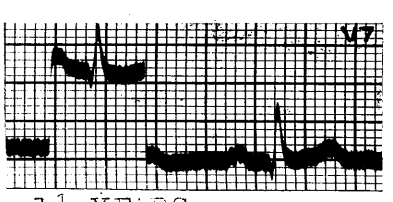












CASE 26, W.A. MALE, 42 YEARS.

5 MONTHS AFTER ONSET.

9 MONTHS AFTER ONSET.

1½ YEARS AFTER ONSET.

A.

COMPARISON of STANDARD LIMB LEADS and UNIPOLAR LEADS in the DIAGNOSIS of ANTEROLATERAL INFARCTION

Of one hundred and sixty-one cases of myocardial infarction analysed by Myers et al (1948b), fifty-seven were anterolateral in situation and of these, twenty-six had diagnostic signs in lead I. Thirty of the total series are described in detail and can be subdivided into sixteen with diagnostic signs in lead I, six with signs of left ventricular hypertrophy in the standard limb leads, and four with flat T waves in lead I which would at least call for further electrocardiographic investigation. The remaining four show no indication of infarction in the standard limb leads; they present patterns of non-specific abnormalities including bundle branch block or of coexistent posterior infarction.

Bain and Redfern (1948) described the electrocardiograms of seven cases of extensive anterior infarction, each case showing diagnostic signs in all chest leads. The T wave of lead VL was inverted in all seven cases but only six of them showed inversion of the T wave in standard limb lead I, including three with inversion in lead II also; in the seventh case, the T wave of lead I was flat.

It /

/ It is noteworthy that only fifteen out of sixty-six cases showing a pattern of T1 less than T3 (both being positive) showed electrographic signs of anterolateral infarction while forty were anteroseptal (Dressler and Roesler, 1948). The authors point out that this pattern only arises when T of the right arm is more negative than T of the left arm. Since in anterolateral infarction, the left arm as a rule displays a marginal or central zonal pattern, and remains abnormal even in the healed stages, the infrequency of the pattern is explained. In a recent article, Fiske (1950) states that lead aVL is less informative than lead I in anterolateral infarction. This statement is based on his finding that in three out of five cases of anterolateral infarction, the Q wave of lead I was greater than that of lead aVL and also that the ratio of Q to R in lead I was greater than that in lead aVL. Besides the T wave tended to be deeper in lead I than in lead aVL. He makes the suggestion "that the R waves of the unipolar limb leads which contribute to the formation of the Q waves of the standard limb leads may be more than mere passive agents." The existence of a myocardial infarction may influence the time of onset of the R wave in, say, unipolar lead VR and thus alter the contour of standard limb leads I and II. The present work lends favour to the thesis that lead VR is often specifically affected in anterolateral infarction.

As /

/ As stated in the introduction, the right arm may be in relation to the endocardial surface of the septal portion of the infarction or to the endocardial surface of an extension of the infarct into the anterior wall of the right ventricle, which is however, uncommon. The resultant signs chiefly affect the ST-T segment and T wave; the ST-T segment becomes depressed and subsequently the T wave becomes peaked; less frequently, the QRS deflection as well as the ST-T segment is directly influenced by the presence of the infarction. These changes are superimposed on the variable physiological contour of lead VR. Hence from the standpoint of lead VR the present series of eleven cases may be subdivided into four groups as follows:

i. lead VR with rS or QS pattern not in apposition to the infarction;

ii. lead VR with rS or QS pattern in apposition to the infarction;

iii. lead VR with QR pattern not in apposition to the infarction, and

iv. lead VR with QR pattern in apposition to the infarction.

i. Lead VR with rS or QS pattern not in apposition to the infarction.

If a small r wave is present in lead VR its reciprocal increases the Q wave of lead VL in the formation of standard limb lead I. The deep S wave of lead VR, in reciprocal, heightens the R wave of lead VL. If lead VR shows a /

/ shows a QS deflection derived from the cavity of the right ventricle, it cancels the Q wave of lead VL for at any given instant the negativity of the right arm is greater than that of the left. In late cases also, where there is a permanent conduction defect in the lateral wall of the left ventricle, indicated by a short broad Q wave and delayed R wave in lead VL, the Q wave is annulled by the immediate and prolonged negativity of the right arm and hence lead I shows only a broad R wave of moderate height. (case 16). Owing to the age of the infarction in such cases, the ST-T signs are usually of non-specific character; the ST-T segment is usually isoelectric and the T wave flat; hence lead I presents no sign of infarction as such, only non-specific abnormalities calling for further electrocardiographic investigation.

As stated above, lead VR more commonly apposes the cavity of the right ventricle rather than its anterior wall and therefore its T wave is more often inverted than upright. However it is rare for lead VR to retain a normal inverted T wave in the presence of any severe infarction even although it does not directly appose it, and thus, in reciprocal, to neutralise the inverted T wave of lead VL. More commonly, the T wave of lead VR is upright and symmetrical and its ultimate effect is to deepen the inversion of the T wave of lead VL when it is transferred to lead I. However, when the infarct is

/ is healed, the T wave of lead VR may again become inverted and therefore militate against the negativity of the T wave of lead VL which is often a permanent sign of healed anterolateral infarction.

If there is a severe degree of forward displacement of the apex, the left arm may face the centre rather than the left marginal zone of the infarct and therefore register a QS rather than a QR pattern while the right arm shows, as above, an rS deflection. The Q wave relationships remain as above but the R wave of lead I is derived entirely from the S wave of lead VR and the continued negativity of the left arm provides an S wave in lead I. (case 17).

ii. Lead VR with rS or QS pattern in apposition to the infarction.

If the infarct is large enough to involve the anterior part of the septum and its terminus in the anterior cardiac wall and if the right arm is in a position to record septal activity, then its initial small r wave is abolished, like that of leads V1 and V2, for there is no longer any activating impulse passing from the left side towards the right in the anterior part of the septum. (case 18). The downward limb of the remaining QS deflection in reciprocal diminishes the Q wave of lead VL so that the final Q of standard limb lead I may be small. (case 19). In late tracings /

/tracings evidence of local healing in the anterior part of the septum may be furnished by the restitution of the early R wave in lead VR; when, however it is transferred, in reciprocal, to form standard limb lead I, it increases any abnormal Q wave in that lead derived from lead VL (case 26). In recent tracings the apposition of lead VR to the endocardial surface of the infarction causes the ST segment to be depressed and the T wave to be diphasic. (case 18). This increases the elevation of the ST segment of lead VL when it is carried into standard limb lead I. Later, the T wave of lead VR becomes peaked and upright and, in reciprocal, increases the negativity of the inverted T wave of standard limb lead I derived from lead VL.

When the infarction is healed there is frequently a conduction defect in the lateral wall of the left ventricle. This causes a broad thickened Q wave in lead VL which is simultaneous with the maximum negativity of lead VR. The result in standard limb lead I is a much less conspicuous or even absent Q wave. (cases 18, 19). In well healed infarctions the R wave of lead VL is frequently tall although slightly delayed. This is carried into lead I more or less unchanged since its peak is later than the nadir of the QS of lead VR. The ST segments of leads VR and VL and therefore of lead I are now iso-electric but the restitution of the normal inverted T wave of lead VR

/ lead VR diminishes the negativity of the residual inverted T wave of lead VL, when it is carried into lead I (cases 18, 19); similarly the persistence of an abnormal inversion of the T wave in lead VR when coupled with a similar T wave in lead VL causes a flat T wave in standard limb lead I. (case 26).

iii. Lead VR with QR pattern not in apposition to the infarction.

It is very probable that the presence of an anterolateral infarction, especially if imperfectly healing, causes the heart to become more horizontal and also pushes the apex posteriorly. The latter movement causes the transitional praecordial pattern, i.e. the change from a right to a left ventricular pattern to be displaced to the left. Hence S waves are found in leads V4, V5 and even V6 and the R waves are less prominent than would have otherwise been expected. The same backward rotation of the apex causes the back of the heart to face the right arm and hence it registers a QR deflection with inverted T wave.

It is an interesting fact that the cardiac position above described occurs only once in the early tracings of fifteen anteroseptal infarctions but it does occur in five out of eleven anterolateral infarctions. When this cardiac position obtains, the Q wave of lead VR in reciprocal tends to neutralise that of lead VL and hence in standard limb lead I it is smaller than in lead VL. If, however, the /

/ the Q wave of lead VL is completed early in the QRS interval it may precede the later Q wave of lead VR and therefore be transmitted with little diminution to standard limb lead I. In such cases the chief effect of the Q wave of lead VR is to enhance the R wave of lead VL (case 20). In either case the R wave of lead VR supplies the S wave of lead I. Slight physiological elevation of the ST segment is not uncommon in lead VR, hence the final ST pattern in lead I may be nearer the iso-electric line than in lead VL. Similarly the T waves of leads VR and VL may largely cancel each other. (case 21).

iv. Lead VR with QR pattern in apposition to the infarction.

As in the previous group the normal deep Q wave of lead VR minimises or obliterates that of lead VL when it is carried into standard limb lead I. (cases 22, 23). The R wave of lead VL is recorded sooner than that of lead VR for the latter is derived from the last portion of the left ventricular wall to be activated, viz. the posterobasal region, hence, as in group iii, the small R wave of lead VL tends to be augmented by the unspent negativity of the Q wave of the right arm, in reciprocal. The late positivity of lead VR accounts for the prominent S wave of lead I. (cases 22, 23).

Because lead VR is in apposition to the infarction the ST segment in early tracings shows downward convexity which /

/ which increases the upward ST bowing in standard limb lead I, when added to that of lead VL. However the S wave of lead I prevents elevation of the ST-T junction. Even at an early date the T wave of lead VR shows symmetrical upright peaking which determines the inversion of the T wave in lead I for at this early stage there may be no inversion of the T wave in lead VL. (cases 22, 23).

In the healed stages of extensive infarctions lead VL usually displays the signs of a local defect of conduction in the lateral wall, as previously stated. The prominent slow Q wave may be contemporaneous with the normal Q wave of lead VR and they may cancel each other in standard limb lead I. (cases 22, 24). The tall delayed R wave of lead VL may be simultaneous with the tall normal R wave of lead VR so that the final R wave of lead I is the difference between those of leads VR and VL. Any remaining positivity of the right arm is registered as an S wave in lead I. ST segment changes are now minimal. The T wave of lead VL is usually inverted; this pattern is transmitted to lead I, either augmented by the persistence of an abnormal upright T in lead VR, or unchanged, if the T wave of lead VR is flat. (case 24).

It is clear from the above that lead VR exerts a variety of effects on a basic diagnostic QR pattern in lead VL. At times the signs of infarction are exaggerated either by the normal deflections of lead VR or by effects /

/ effects specifically derived from the infarction, but more often, important signs may be mitigated or obscured in the final pattern of lead I. In view of the heterogeneous origin of the various deflections of lead I it is clearly necessary to analyse and evaluate them by reference to leads VR and VL.

Occasionally lead VL, as stated above, registers a QS deflection instead of a diagnostic QR deflection. This may occur if the electrode on the left arm is apposed to the central zone of infarction on account of forward rotation of the cardiac apex or clockwise rotation of the heart around its own long axis. The QS may be short and blunt, (case 17); its association with upward convexity of the ST segment is further proof of its abnormal origin. However a deep QS deflection in lead VL is a normal finding in vertical hearts because the left arm lead as well as the right arm lead face into the ventricular cavities and hence record cavity potentials. Being thus at right angles to the plane of any infarction in the anterior or lateral wall, the left arm electrode does not record changes specifically arising in the infarction, e.g. ST changes. However it may record changes arising in the peripheral zone of ischaemia, viz. cove-plane T waves. Somewhat different appearances may be presented by lead VR in vertical hearts with anterior infarction for the lead on the right arm may face, however obliquely, the endocardial surface of the infarct if it /

/ it extends into the septum or into the right ventricle and thus may record depression of the ST segment, followed by positivity of the terminal part of the T wave and finally reversion to the normal inverted T wave of lead VR. The above features are seen in case 25. The deep QS deflections of leads VR and VL largely cancel each other and hence the QRS deflection of lead I is very small and of little diagnostic value. However lead I shows elevation of the ST segment which is the reciprocal of the depression of the ST segment in lead VR for it is iso-electric in lead VL. The subsequent large cove-plane T waves of lead I are directly derived from those of lead VL. Isolated ST-T changes in lead I are insufficient for the complete diagnosis of anterior infarction since they may occur in pericarditis. The failure of diagnostic QRS signs in this case is due to the minuteness of the QRS complex which is in turn due to the very vertical position of the heart.

Thus of the eleven cases of anterolateral infarction five show diagnostic signs of infarction in standard limb lead I (cases 17, 18, 19, 22, 26), viz. the presence of a Q wave, a small R wave, some elevation of the RS-T junction in early tracings, upward convexity of the ST segment and, in later tracings, inversion of the T wave. All five have intermediate to horizontal hearts. Two further cases (Nos. 23, 24) show signs in lead I very suggestive of infarction but their failure to be diagnostic is due to /

/ to the absence of a Q wave, the original significant Q wave of lead VL being cancelled out by simultaneous negativity of the right arm due to its facing the back of the heart. However one of the two cases (No. 23) subsequently developed an extension of the infarction more deeply into the lateral wall with increase of the Q wave and diminution of the R wave of lead VL. These changes are transferred to standard limb lead I which now shows a small Q wave and very short R wave. The other case (No. 24) is similar to the above in showing no Q wave in standard limb lead I; there is however upward convexity of the ST segment and a large inverted T wave. Two cases show equivocal QRS changes in standard limb lead I; in the first (No. 21), because of the tendency for lead VR to mitigate the diagnostic signs of lead VL and in the second (No. 25) because of the very vertical position of the heart but both show ST-T changes very suggestive of anterior infarction. The remaining two cases (Nos. 16, 20) show non-specific abnormalities in standard limb lead I partly due to the age of the infarct but the flat ST-T formation would certainly have raised the suspicion of a previous anterior infarction.

The higher proportion of diagnostic signs in standard limb lead I in anterolateral compared to anteroseptal infarction is to be expected in view of the close relation--ship of the left arm lead to the lateral ventricular wall. As a rule the left arm derives its potentials from the V5

/ V5 level which is typically involved in the lateral part of the infarction, although as explained above, the signs of infarction recorded by lead VL are exaggerated or mitigated by the reciprocal of lead VR. As in the previous series of anteroseptal infarction, diagnostic signs may be absent in lead VL, and therefore in lead I, if the infarct is of long standing, since ST-T abnormalities tend to lessen with age. Furthermore, left ventricle hypertrophy with horizontal cardiac position is prone to develop in the healed stages of infarction. The associated counterclock--wise rotation makes it all the more improbable that lead VL, and hence standard limb lead I will derive its potentials from the scarred region of the heart.

The ratio of T1 to T3 requires brief comment. Of the ten cases of anterolateral infarction, only two show patterns of T1 less than T3 but in only one of them is it attributable to the presence of a healed anterolateral infarction, one year after onset. In the other case it can be explained by the vertical position of the heart, the residual signs of infarction being limited to the anterior praecordial leads. Conversely, in a third case, the T wave of standard limb lead I is more positive than that of lead III although the chest leads show the signs of a healed anterolateral infarction. In these circumstances the value of the T1/T3 ratio as an index of a focal myocardial lesion is very doubtful.

SUMMARY

An analysis has been made of standard limb lead I in eleven cases of anterolateral infarction. Five showed diagnostic signs and four others very suggestive signs of infarction in this lead. While the form of lead VL is usually derived from the marginal zone of infarction, i.e. it shows a significant QR deflection, that of lead VR is variable according to its relationship to anterior right ventricular wall and cavity and posterior cardiac wall on the one hand and to the endocardial surface of extensions of the infarction into the septum and right ventricular wall on the other. While lead VR generally tends to mitigate the diagnostic signs of lead VL, it may also augment them either by the addition of its own physiological configuration or by effects actively and specifically derived from the infarction. Thus the heterogeneous origin of the various deflections of standard limb lead I makes it necessary to analyse them by reference to leads VR and VL. The frequency of horizontal position with backward rotation of the apex is noted in the healing stages of anterolateral infarction; it is suggested that the latter may encourage the former.

It is in this position of the heart that the /

/ the mitigating effect of lead VR on the diagnostic signs in lead VL is particularly conspicuous. If the heart assumes a horizontal position with counterclockwise rotation, due to left ventricular hypertrophy, or if the infarction is of some standing, standard limb lead I may fail to show diagnostic signs.

The pattern of T1 less than T3, both being positive, is of doubtful value as an indication of an anterolateral lesion.

B.

UNIPOLAR ELECTROCARDIOGRAPHY AS AN AID TO DETERMINING SITE, EXTENT and HEALING OF ANTEROLATERAL INFARCTION.

The diagnosis of the lateral extension of infarction, which is the differentiation between anteroseptal and anterolateral infarctions, rests on the presence of diagnostic signs in leads V5 and VL or in leads V5 and V6. Of fifty-seven anterolateral infarctions described by Myers et al (1948b), only fifteen show QS or abnormal QR deflections in leads V5 and either VL or V6, whereas twenty-seven show significant signs in all three leads. Such a high incidence of signs in all three leads in a series of cases all of which came to autopsy would favour the view that the prognosis is worse the greater the number of leads affected. Myers found a QS deflection in lead V5 or in leads V5 and V6 in twenty-four instances, and, of the twenty-four, twelve showed transmural infarction of the apical one third or more of the lateral wall of the left ventricle. Similarly he found an abnormal QR deflection in the same leads in the remaining thirty-three cases of the total series and of the thirty-three, twenty-seven showed subendocardial infarction of the lateral apical wall. The anteroseptal portion of the lesion, as opposed to the lateral part, showed the

/ the following correlation. Of the total of fifty-seven cases of anterolateral infarction, thirty-eight had a transmural infarction of the apical one third of the anteroseptal wall, of whom twenty-nine showed QS deflections in leads V3 and/or V4. Nineteen of the fifty-seven had a subendocardial infarction of the same region and, of them, eleven showed abnormal QR or very coarsely notched QS complexes in these leads. Myers also found that typical and sequential RS-T changes usually accompanied QS deflections in the praecordial leads, indicating injury to the subepicardial muscle; similar though less severe appearances were found in association with recent subendocardial infarction and, presumably, indicate minor subepicardial damage, but, if the lesion is strictly limited to the subendocardial layer, the RS-T segment is depressed. When ventricular aneurysm develops he found that the RS-T changes became permanent.

The seven cases of extensive anterior infarction described by Bain and Redfern (1950), showed a QS deflection, elevation of the RS-T junction and deep inversion of the T wave in all six praecordial leads.

There are eleven examples of anterolateral infarction in the present series; nine have electrocardiograms taken within two months of onset (i.e. all cases except Nos. 24 and 26), and, of the nine, eight show QS or significant QR deflections in

/ in leads V5 and VL and one in leads V5 and V6 (case 25). Of the eight cases showing diagnostic signs in leads V5 and VL, seven, (i.e. excepting No. 17), show a QR, not a QS complex, in lead VL indicating that the infarction has been confined to the subendocardial layer of that part of the lateral wall of the left ventricle subtended by the electrode on the left arm. Alternatively the R wave may have been derived from intact portions of the lateral wall. The eighth case (No. 17), shows a QS deflection in lead VL, the heart being in such a position that potentials from the central zone of infarction have been referred to the left arm.

Of the remaining two cases of the original list of eleven (Nos. 24 and 26) the electrocardiograms of which were not obtained until some months after onset, one, viz. No. 26 shows a significant QR deflection in leads V5, V6 and VL in the first instance. The other, No. 24 was a case of anterolateral aneurysm clinically and radiologically. The absence of a Q wave from leads V5 and V6 should strictly speaking, have excluded this case from the anterolateral group but the low delayed R waves of leads V5 and V6 along with the conduction defect recorded by lead VL strongly favour the view that the lateral wall is involved.

While unequivocal QRS signs are present in leads V5 and VL in ten cases, irrespective of age of infarct, only /

/ only borderline or non-specific abnormalities are seen in lead V6, viz. diminution in height of the R wave, elevation of the ST segment and, later, inversion of the T wave. The rarity of a QS deflection in lead V6 is noted by Myers who found that even if the lateral wall shows transmural infarction, it is only the apical one third which is affected and hence an R wave is invariably provided by unaffected regions. Case No. 25 is somewhat atypical in that while a small abnormal QS complex is present in lead V5, there is no Q wave in lead V6 and only a small R wave is present. There are however, conspicuous ST-T changes. While these early signs in lead V6 may be explained by pericarditis, the later development of a prominent R wave in lead V6 (as well as in leads V5 and V7) suggests that the lateral wall has originally been involved in the infarction, although even this sign may occur in pericarditis. The present series contains several examples where there is conspicuous diminution in the height of the R wave in the axillary leads suggesting involvement of the lateral wall. However quantitative reduction can only be confirmed in retrospect, if with healing, the R wave increases in height (cases 18, 19, 20, 25, 26). The effect of cardiac position in causing small R waves in the axillary leads is discussed later.

The typical pattern of anterolateral infarction is /

/ is also characterised by QS or abnormal QR complexes in leads V3 and V4 as well as in leads V5, V6 and/or VL. Eight cases with early tracings show QS deflections in lead V4 and all but one, (case 19), in lead V3 also. Myers notes that the incidence of a QS in both leads V3 and V4 is higher in anterolateral than in anteroseptal infarctions - a fact borne out by the present group. Absence of the R wave occurs in leads V1 and V2 in six out of the eleven cases and may indicate extension of the anterolateral infarction into the anterior terminus of the septum, (cases 18, 21, 22, 23, 24, 25). This interpretation is justifiable if the heart is vertical and therefore the potential variations of the ventricular cavities are unlikely to be referred to the V1 and V2 positions (case 25), or if, on healing, an R wave reappears in these leads (case 22). Otherwise absence of the R waves in leads V1 and V2 may not be pathological since it is a normal finding in some horizontal hearts, especially in the presence of left ventricular hypertrophy. Involvement of the septum is suggested, in retrospect in case 26 by the reappearance of the early R wave of lead VR the original ablation of which was probably due to septal involvement but the persistence of an unaffected R wave in lead V1 militates in some measure against this view.

Changes in the ST-T deflections in the present series conform to the usual pattern. There is elevation of the /

/ the ST-T junction and upward convexity of the ST segment with monophasic upright T wave in early cases. These changes are greatest in leads V2, V3 and V4 (cases 18, 22, 23, 25). Although the associated QS deflection indicates a transmural infarction this ST-T pattern indicates the presence of subepicardial tissue which is injured but not dead. Frequently ST changes of minor degree occur as far to the left as lead V6 or even V7 although there is no longer a significant Q wave; the R wave however is usually diminished in height (cases 24, 25, 26).

One case demonstrates the combination of ST-T changes of recent type in leads V4 and V5 in association with a QRS pattern indicative of a healed anterolateral infarction. As discussed in the case report, the ST-T signs are probably due to acute pericarditis complicating such an infarction (case 16).

Inversion of the T wave following the return of the ST-T junction to normal levels affects most of the praecordial leads in some measure. Leads V4 and V5 show the deepest inversion, leads V6 and V7 lesser degrees of it (cases 17 to 22). In vertical hearts, inversion of the T wave is deepest in leads which have shown the greatest elevation of the ST junction, viz. in leads V3 and V4, (case 25), but in other cardiac positions the deepest T waves are generally seen in the lead to the left of /

/ of that showing the highest ST elevation (case 22). Changes in the T waves may be minimal in leads over the right side of the praecordium, viz. transitory inversion of the end of the T wave (case 22). Where leads V1 and V2 are facing the ventricular cavities, they may register T waves partly derived from the endocardial surface of the extension of the infarction into septum or anterior wall of right ventricle, viz. sharply upright T waves, comparable to those of lead VR (case 22).

The changes in the ST-T deflections may persist in minor degree for an indefinite period - as shown by all eleven cases of the present series. Slight elevation of the ST junction with upward convexity of the ST segment is seen in lead V4 nine weeks after onset in case 17 and one year after onset in case 21. In the case of anterolateral aneurysm (case 24), it is seen in a lead over the aneurysm as well as in leads V2, V3 and V4 nine months after onset of illness. Persistence of an elevated ST segment and monophasic upright T wave in ventricular aneurysm has been described by Wilson (1944) and by Myers (1948b). In leads further to the left the ST segment may be isoelectric but its shape may show persistent upward convexity with inversion of the T wave whereas in leads over the right side of the praecordium the ST take-off may remain two to three millimetres above the base line. However this level may not lie beyond /

/ beyond normal limits for these leads and furthermore the ST segment frequently shows a normal contour, viz. an upward concavity, in later tracings when it is also accompanied by an upright T wave. Nevertheless this ST-T formation may be accompanied by a persistent QS deflection. The association of this pattern with old healed transmural anterolateral infarction was noted by Myers (1948b).

Spread of infarction may be diagnosed if diagnostic signs appear in a lead in which they were previously absent. Case 23 shows increased elevation of the ST junction and increased upward convexity of the ST segment with deepening of the Q wave and diminution of the R wave. These changes occur two weeks after onset and affect leads VL and V5. For the first time also, leads V6 and V7 show slight elevation of the ST segment and diminution in the height of the R wave. While these changes indicate extension of infarct in the lateral ventricular wall, signs of healing are seen in leads over the right side of the praecordium. The difficulty of assessing the size of an infarct by reference to the number of leads showing diagnostic abnormalities is well recognised. In their correlation of electrocardiographic and pathological signs, Myers et al (1948b) found that a central zonal pattern, limited to leads V3 and V4, might occur with extensive anterior lesions as well as with infarctions limited to

/ to the apical one third of the anterior wall; similarly a QS or an abnormal QR pattern might be found in all three leads, V5, V6 and VL, although the infarct was confined to the apical one third of the lateral wall, whereas a large infarction involving half of the lateral wall might be documented by abnormalities in only one or two leads. Furthermore, Myers points out that if the thorax and heart are small, an infarct of given size tends to produce QRS abnormalities in a greater number of leads. Another factor which may account in large measure for these anomalies is the electrical position of the heart; when the heart is vertical and the plane of the anterior infarction is parallel to the chest wall, QS or significant QR deflections occur in the maximum number of praecordial leads, although not in lead VL (case 25). Other positions of the heart are more common and various rotational combinations occur. While the typical pattern of anterolateral infarction as indicated above is frequently presented, it may be engrafted on a less common but more obtrusive electrocardiographic pattern derived from the electrical cardiac position. For example five of the eleven cases (20, 21, 22, 23, 24) have a horizontal or semihorizontal heart with backward rotation of the apex and only a lesser degree of counterclockwise rotation around its own long axis. The sum total of these changes is to cause the transitional ventricular pattern to be

/ be shifted to the left so that in the apical and axillary leads the R wave remains small and the S wave persists. At the same time a pattern derived from the back of the heart is registered by the right arm. It is possible that the presence of a large anterolateral infarction, especially if healing poorly, may affect the electrical (and perhaps also the anatomical) position of the heart and encourage it to assume the above position.

These considerations are also of importance in the later months when patterns of healing are developing. These are partly derived from the persistence of early signs and partly from the development of new signs. Of the former, deep QS deflections may become permanent in several praecordial leads, in leads V3 and V4 (case 17) or in leads V1 to V4 inclusive (cases 18, 20, 21, 22, 24). In other cases lead V4 may show signs of regeneration in the development of a late R wave indicating that, although there was originally a transmural lesion as seen by a QS deflection, the subepicardial muscle cannot have been totally destroyed: sufficient vitality has survived to provide a basis for regeneration. In vertical, semivertical or intermediate hearts, this R wave forms a conspicuous upward deflection following the prominent Q wave in lead V4 or in V4 and V5 (cases 16, 19, 25). Sometimes a similar but smaller R wave may develop in lead V3 at a later date (case 19). The /

Note : Following data found in [illegible]

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/ The axillary leads may also show a further late increase in height of their R waves along with shelving ST segments and persistently inverted T waves. At the same time the deep QS deflections of leads over the right side of the praecordium lengthen so that the final picture is indicative of some degree of left ventricular hypertrophy (cases 18, 19).

In horizontal or semihorizontal hearts with the rotational position indicated previously, regeneration is also shown by the development of an R wave in lead V5 where previously there was a QS deflection but it precedes the deep S wave (case 22). In lead V4 it is represented by shortening and splintering of the previously deep QS deflection. The R wave of the axillary leads V6 and V7 shows little increase in height but leads VL and VF are more advantageously placed when the apex of a horizontal heart is rotated backwards to detect the increase in left ventricular function which does take place. Lead VL shows a much heightened R wave and lead VF a deepened S wave. These cases demonstrate the important part played by the electrical cardiac position in determining the late electrocardiographic pattern, although the pathological substrate of early left ventricular hypertrophy or dilatation along with laminar functional recovery at the left margin of the infarct may be the same.

The /

/ The signs in lead VL are somewhat less dependent on cardiac position. The commonest residual sign is a short truncated Q wave with some delay in attaining the peak of the tall R wave which follows. These signs indicate a conduction defect in the lateral wall of the left ventricle. (cases 16, 18, 19, 22).

SUMMARY

Eleven cases of anterolateral infarction have been analysed. A QS or significant QR deflection was found in leads V5 and VL in eight cases, in leads V5 and V6 in one case and in all three leads in one case. The eleventh case (that of anterolateral aneurysm) was atypical in the disposition of diagnostic QRS abnormalities. Of the eight cases, seven showed a QR complex in lead VL and one a QS complex. As a rule lead V6 showed only nonspecific abnormalities but temporary diminution in the height of the R wave occurred in several cases. All eleven cases presented QS deflections in lead V4 and ten in lead V3 also. The significance of the absence of the R wave in leads V1 and V2 which occurred in six cases is discussed. Early changes in the ST-T segment were greatest in leads V2, V3 and V4 although inversion of the T waves reached its /

/ its maximum in leads V4 and V5. The persistence of ST-T changes was noted especially in the case of anterolateral aneurysm.

Signs indicating spread of infarction farther into the lateral wall were presented by one case.

It is important to recognise the role played by the electrical position of the heart in, firstly, determining the number of leads likely to show diagnostic signs and, secondly, in shaping the basic pattern of the unipolar leads upon which the signs of infarction are superimposed. The presence of a large anterolateral infarction probably causes a change in the electrical (and perhaps also the anatomical) position of the heart. The final patterns of healing are determined by the combination, in varying degrees, of the persistence of the early signs of infarction, and of the development of new signs. In the first group, deep QS deflections may persist especially in leads V3 and V4. In the second group new R waves may develop in lead V4 or the axillary leads may ultimately display the signs of increased left ventricular function. Signs of a localised conduction defect in the lateral ventricular wall are not infrequent in lead VL in the later months after infarction.

SECTION V

POSTERIOR INFARCTION

SECTION V

POSTERIOR INFARCTION

INTRODUCTION

The difficulty surrounding the diagnosis of posterior myocardial infarction has long been recognised. Standard limb lead III has very wide physiological variation, some of which simulate the signs of posterior infarction. As indicated in the historical review (p.11) Pardee (1930) set up criteria to differentiate a normal from an abnormal Q wave in lead III but later workers found that his standards still included non-infarcted hearts. Diagnosis based on the unipolar left leg lead was much more accurate when checked against oesophageal leads (Myers, 1949c) or finally by postmortem examination. However some of the normal vagaries of lead VF mimic closely the pattern of infarction so that it became essential to establish standards for the recognition of an abnormal Q wave in this lead. The criteria of Goldberger (1945) and of Myers (1949c) have been discussed in the historical review (p.51). The latter have largely been adopted in the following pages and will therefore be summarised here. For the diagnosis of posterior infarction the QR complex of lead VF should measure 0.5 millivolt, the Q wave should last 0.03 seconds or more from onset to nadir and should exceed 25 per cent of the succeeding R wave. Such a pattern indicates

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/ indicates posterior infarction. Frequently only one of the two criteria for the Q wave itself are fulfilled; in such instances the QR complex is classed as borderline to strongly suggestive. If the QR complex is less than 0.5 millivolts the pattern is considered to be strongly suggestive of infarction if the two criteria with respect to the Q wave itself are fulfilled, and diagnostic, if the Q wave is specially prolonged, viz. 0.04 seconds. In healed infarctions the pattern of a conduction defect in the posterior wall is frequently seen in lead VF. It has the following characteristics the QR complex should measure 0.5 millivolt or more, the upstroke of the R wave should be prolonged, notched or coarsely slurred but the duration of the Q wave and the Q/R ratio are less important.

Difficulty arises in differentiating the normal QS deflection found in lead VF in horizontal hearts with that due to infarction of the posterior half of the septum. If it is accompanied by a characteristic RS-T pattern, the balance is in favour of infarction. Similarly, if a second tracing obtained after a change of posture or during deep inspiration causes lead VF to assume a frankly abnormal form, viz. a small Q, a small R and a deep S or a QR complex as previously described, then the evidence would again point to infarction.

Besides cardiac position the exact localisation of the infarct in the posterior wall is a strong determinant of the presence or absence of abnormal signs in lead VF. Signs of /

/ of the infarct are most likely to occur if the middle third of the wall is involved and least likely to occur if the lesion is restricted to the basal one third.

Myers et al (1949c) checked their criteria for the abnormal Q wave in lead VF against oesophageal leads as discussed in the historical review (p.51) and they also obtained oesophageal leads in five of their series of one hundred and ten cases of posterior infarction proved pathologically. In all five cases the oesophageal leads were purely confirmatory of the diagnosis. In the last decade the attitude of cardiologists has altered with regard to oesophageal leads. When first made popular by Hamilton and Nyboer (1938) it was hoped that they would eliminate errors in the diagnosis of infarction of the posterior wall because of their close proximity to it and Nyboer (1941) demonstrated their diagnostic value in this lesion. However, increasing usage has revealed some inherent disadvantages; e.g. the oesophagus may be in relation to the right and not the left ventricle; the normal pattern of the transition zone between auricular and ventricular levels is similar to the pattern of infarct and the technical procedures may present difficulties. Furthermore from the point of view of diagnosis the oesophageal leads have no advantage over lead VF, the findings of which they merely confirm (Oram et al, 1951; Bain, 1951).

The value of the left thigh lead in the diagnosis of

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/ of posterior infarction is still sub judice on two points; how frequently does lead VF clarify the diagnosis of posterior infarction which on the basis of the standard limb leads is doubtful, and, secondly, are leads VR and VL specifically affected by posterior infarction and, hence, may the summation of leads VR, VL and VF in the standard limb leads be more informative than lead VF alone? These points have been considered in the following pages. Recently, Meyer (1949) has stressed the importance of comparing the size of the Q wave of lead III with that of lead VF. If the standard limb leads are concordant and if leads II and III show doubtfully significant Q waves, posterior infarction may be diagnosed if the Q wave of lead VF is smaller than that of lead III but if the standard limb leads are discordant, the width of the Q wave of lead VL is more significant of infarction than its depth.

The present writer has included posterolateral infarction along with posterior infarction in general chiefly because only three (Nos. 36, 37 and 38) of the total of twenty-eight cases show signs in the axillary leads, but not in lead VL, indicating extension of the central zone of infarction into the lateral wall but there are several cases with equivocal signs in the axillary leads. Probably more frequent tracings would have shown that they also are originally posterolateral. The distinction may depend to a considerable extent on cardiac position.

Case 27. E.M., male, 42 years.

This case displays the usual signs of posterior infarct in the standard limb leads taken one day after its occurrence; lead VF is confirmatory. The elevation of ST in leads II and III and VF is reciprocated in the depression of ST in all the praecordial leads. It is very slight in lead V1; it increases through leads V2 and V3 reaching a depth of 2 mm. in lead V4. It again reaches the base line through leads V5 and V6. Of all the praecordial leads lead V4 would appear to be nearest to the infarct in this case, facing its endocardial, not its pericardial, surface. In these electrocardiograms also, T is still upright, but not tall, in leads II, III and VF and it is still of normal height in the praecordial leads.

By the end of the first week, while ST elevation is still at its maximum in leads II, III and VF, the reciprocal depression in the praecordial leads is disappearing from lead V1 inwards and to a less degree from lead V6 inwards. It is still 2 mm. deep in lead V4. By the end of the first week also the high peaked T waves typical of posterior infarction have appeared in the praecordial leads, the peak being greatest in leads V2 and V3. Similar high T waves develop in lead VL. This lead initially showed ST depression. Its prominent R wave and early intrinsic

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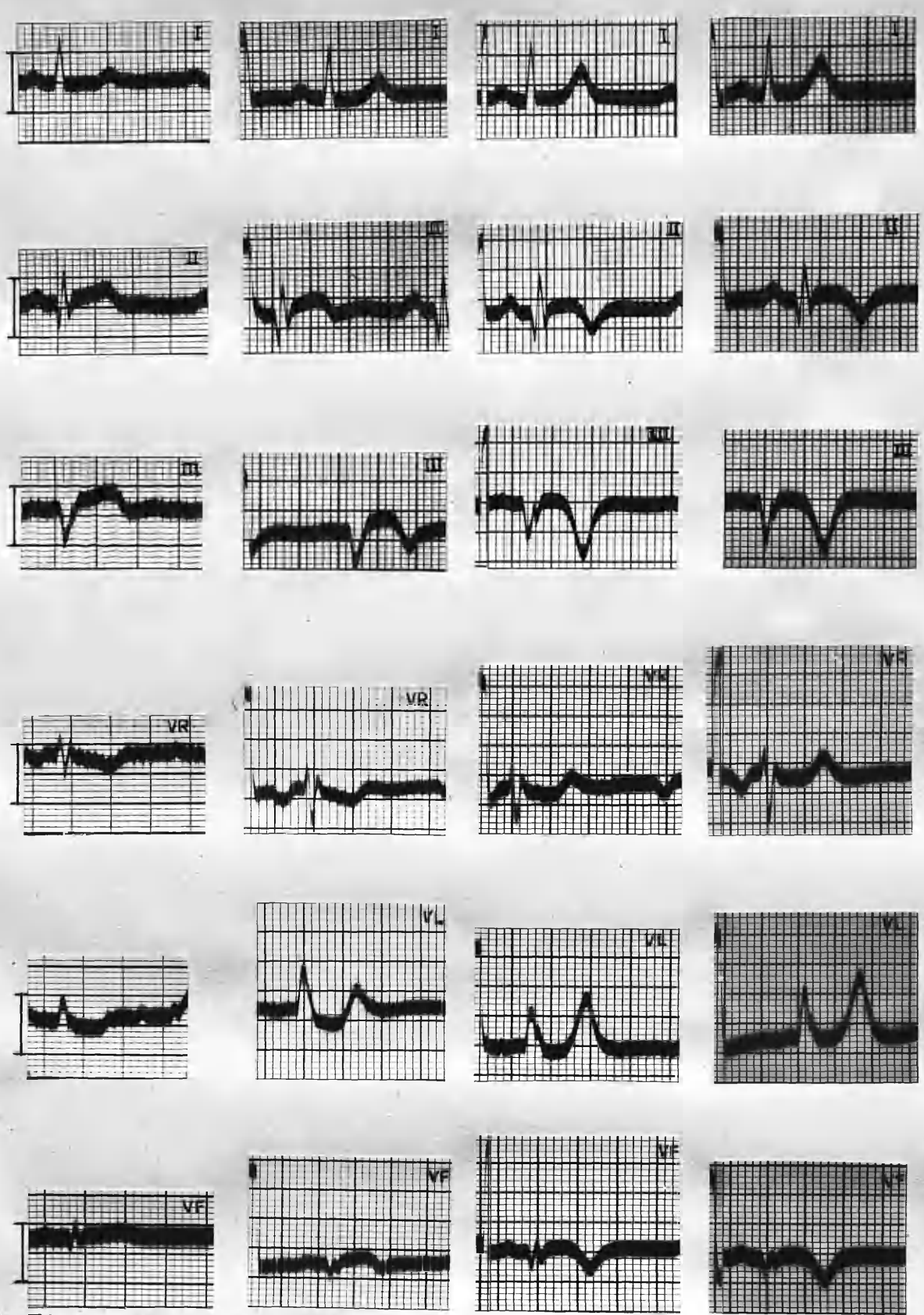
/ intrinsic deflection indicate that it derives its potential variations from the free lateral wall of the left ventricle although the broadening and splintering of the R wave do point to interference in contractility of adjacent left ventricular muscle. Though uninfarcted itself, the potential variations of the left arm are affected, largely but perhaps not entirely in reciprocal fashion, by the presence of the infarct in the posterior wall.

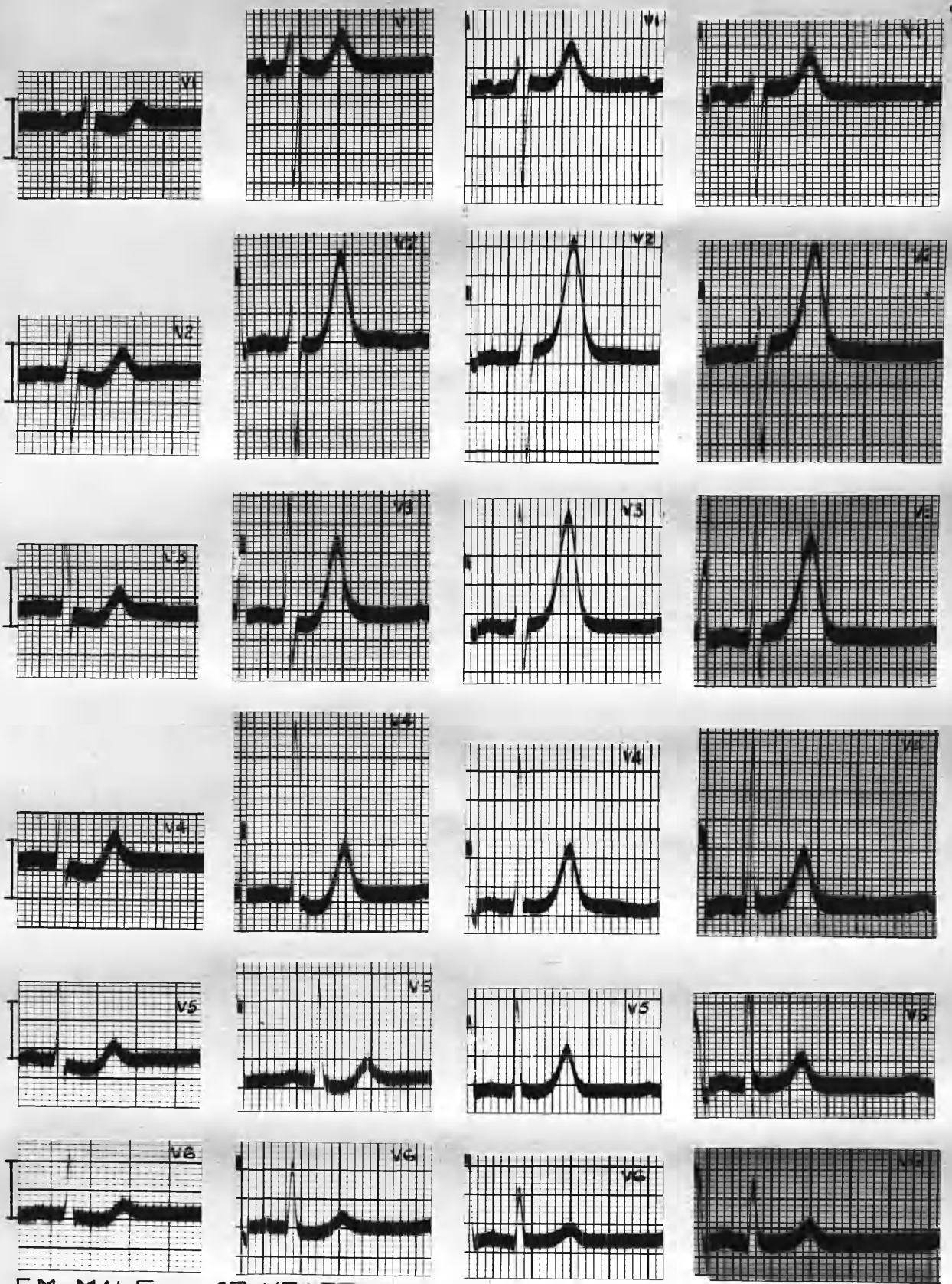
A survey of the electrocardiograms from one to three weeks demonstrates significant R changes in lead V1 and V2. In both, R progressively diminishes in height while it remains a tall deflection in leads V3 and V4. It is well known that a posterior infarction tends to reduce forces that are antagonistic to those which produce the R wave in praecordial leads. The evolution of R demonstrated by this case is a reversal of this process and is to be regarded as a favourable regressive sign indicating even at this early date some functional recovery of muscle at the edge of the infarct. The local pathological substrate may be ischaemia severe enough to interfere temporarily with contractility of muscle, but insufficient to cause any permanent damage; by the end of three weeks this ischaemia has been mitigated to an extent sufficient to restore the normal response to the activating impulse, but not enough to influence the /

/ the disturbance of repolarisation evident reciprocally by the large peaked T waves. Since it is the R wave which is affected in leads V1 and V2, the muscle concerned has been activated early in the RS interval and hence is probably situated in the apical region of the free wall of the left ventricle and, since the potentials are negative in respect of leads V1 and V2, it is likely that the zone of recovery was in the postero-apical region. Such a focal evolution of R being derived from a strictly localised development and occurring only in the V1 and V2 positions is not reflected in the standard limb leads nor in leads VL and VF. Lead VR, however, shows some increase of S. Such increased negativity in a lead, the potentials of which are derived from the ventricular cavities, may indicate a similar return of function in some part of left ventricular muscle. No corresponding R changes are found in outlying leads over the left side of the praecordium.



No 27, E.M., MALE, 42 YEARS.

1 DAY AFTER ONSET.	1 WEEK AFTER ONSET.	2 WEEKS AFTER ONSET.	3 WEEKS AFTER ONSET.



27, E.M., MALE, 42 YEARS.

1 DAY AFTER ONSET.

1 WEEK AFTER ONSET.

2 WEEKS AFTER ONSET.

3 WEEKS AFTER ONSET.

Case 28. J.A., 52 years, male.

This man had a typical attack of coronary thrombosis two and a half months before the first electrocardiogram. Blood pressure was 134/90 and the cardiac sounds were pure. Lead VF is characteristic of a posterior infarction - there is a deep Q wave, a very small R wave, upward bowing of the ST segment and a sharply inverted T wave. The small R wave may be explained by the survival of some muscle elements in the area of left ventricular wall subtended by the electrode of the left leg, presuming the heart is intermediate in position. The tall R in lead VL is found in intermediate, semi-horizontal and horizontal hearts; if the electrical position in this case is either semi-horizontal or horizontal then the appearances in lead VF must be ascribed to infarction of the posterior part of the septum. That such is not the probable electrical position of the heart is suggested by its anatomical position as seen in the X-ray miniature where it is clear that the heart tends to the vertical much more than the horizontal. Furthermore, a cardiac aneurysm developed in this case and its paradoxical pulsation was clearly seen in the postero-diaphragmatic aspect of the apex of the left ventricle.

There is little doubt therefore that the findings in lead VF are due to a transmural infarct of the free posterior wall, the heart being intermediate in electrical position. At this date, two and a half months after onset, reciprocal changes viz: exaggeration of the R and T waves, are striking in the anterior praecordial leads. The S waves are of normal depth in these leads for a heart in the intermediate position. Leads V1, V2 and V3 are closely alike indicating some parallelism between the path of the electrode, the interventricular septum and the area of infarction. The axillary leads are normal.

After the expiry of another two months, the only striking change is the return to normal exhibited by leads V1 and VR in both of which the R and T waves have become reduced in height; in the case of lead VR the T wave is now negative. It is clear therefore that not only does lead VR share in the exaggeration of the R and T waves reciprocal to the presence of a posterior infarction but also that with healing of the infarct the forces normally antagonistic to the R and T waves have become re-established in that particular part of the infarct which influences the V1 and VR leads. It is to be noted that the S waves are not influenced by these

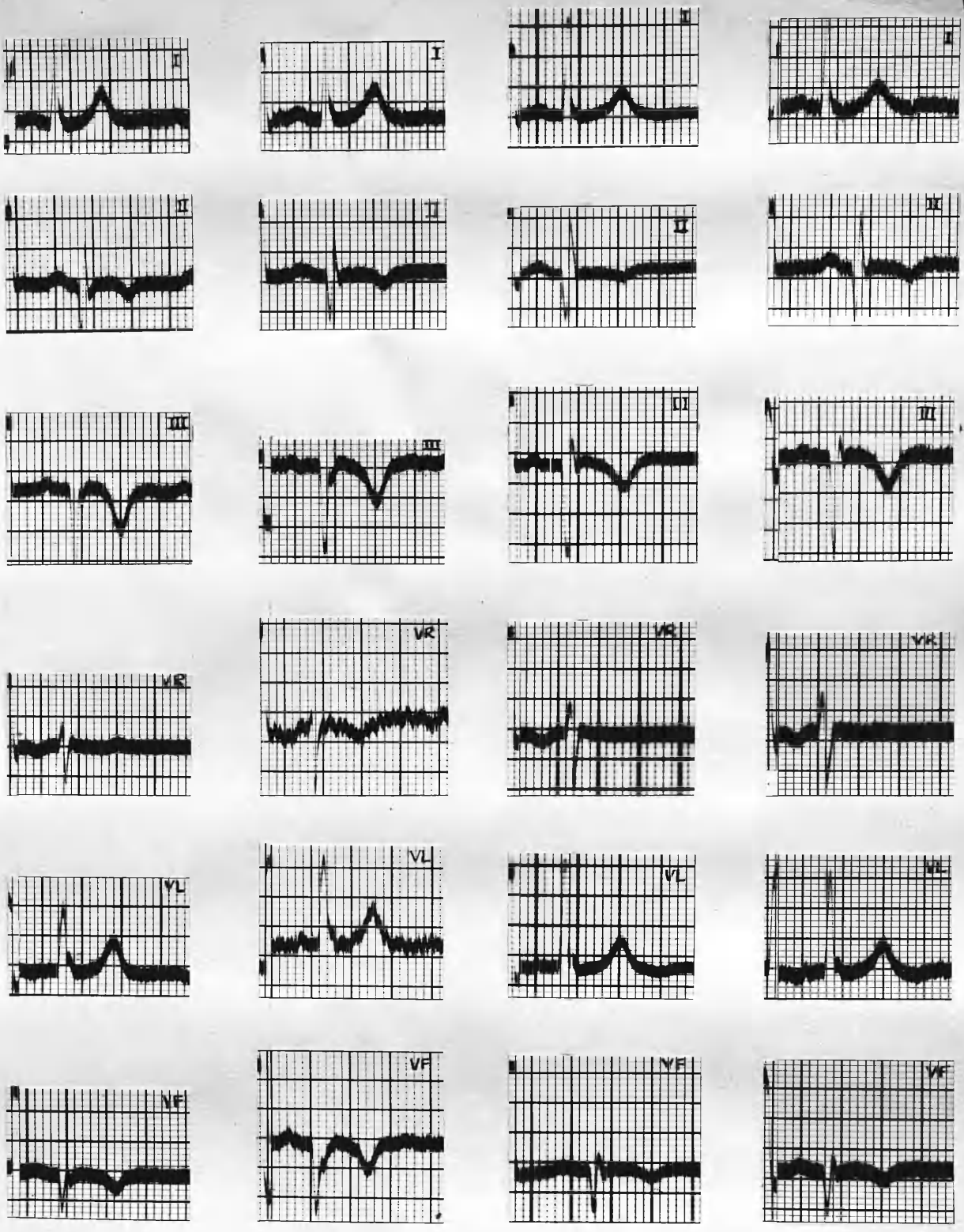
local changes. However, this apparent improvement is ill-sustained since fourteen months later, leads V1 and VR have both reverted to their previous form. In common with these leads, the R and T waves of lead V2 and V3 remain abnormally tall and there is an actual increase in height of the R waves in the apical and axillary leads. Secondarily to this further increment, the S waves of all the praecordial leads from V1 through V5 are lengthening. In addition, the ST segments are sagging and the T waves are becoming diphasic in leads V7 and V6; hence the sudden lowering of the T wave in leads V5 and V4. These ST and T signs are strongly suggestive that the further R and S changes are due to early left ventricular hypertrophy.

The same general appearances are seen in the electrocardiogram taken one year and nine months after onset. They confirm a detail of the T waves of leads V4 and V5 first seen in the preceding series, viz. asynchronous T waves. On the downstroke of the original T wave is a second positive wave. It is clearly not the U wave which is also seen in its usual position in these tracings. No doubt asynchronous repolarisation of the left ventricular wall is the basis of this double T wave.

As stated previously, radioscopy reveals

"paradoxical pulsation in the postero-diaphragmatic aspect of the apex of the left ventricle." With such a total ablation of local potential variations, there are probably no forces antagonistic to the development of the R and T waves over the right side of the praecordium. They are presumably derived entirely from the anterior cardiac wall and septum. The later RST changes in the apical and axillary leads and the S changes in the anterior leads indicate full functional activity of the remaining healthy portions of the left ventricular wall to the stage of actual hypertrophy.

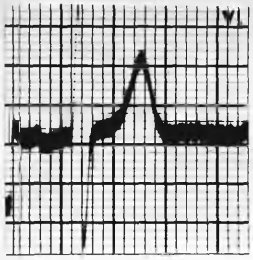
This patient made a good clinical recovery. He did not confess to any anginoid pain and blood pressure remained normal. He resumed work as a cabinet-maker. Five years after his original attack he died of an anterior infarction. There was no post mortem examination.

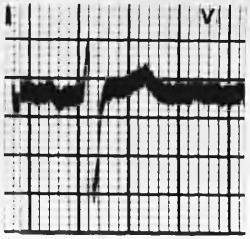


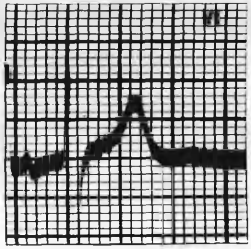
CASE 28,

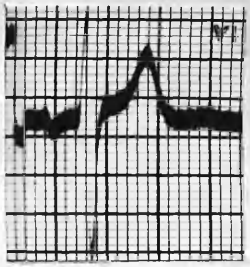
J.A. MALE, 52 YEARS.

2½ MONTHS AFTER ONSET.	4 MONTHS AFTER ONSET.	1½ YEARS AFTER ONSET.	1¾ YEARS AFTER ONSET.


V1


V1


V1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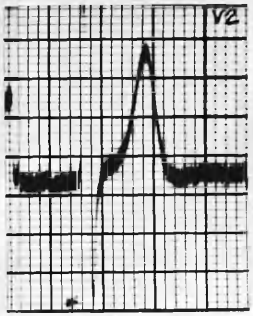
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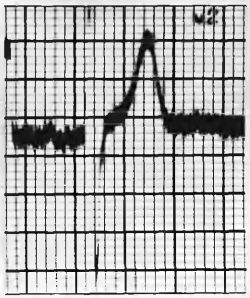
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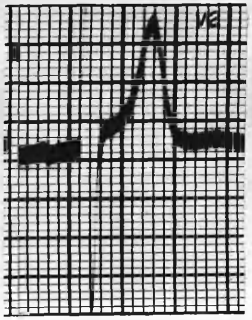
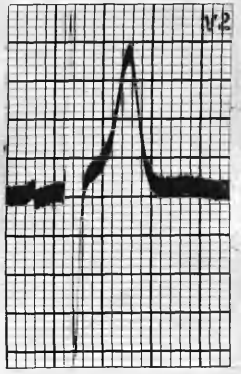

V2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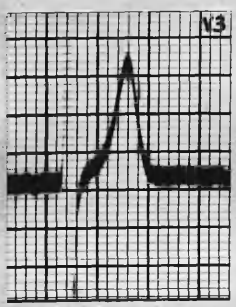
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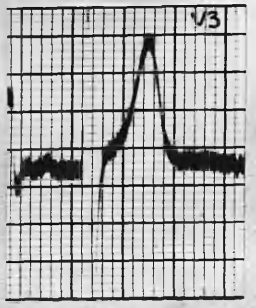
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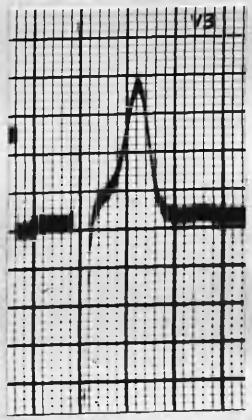
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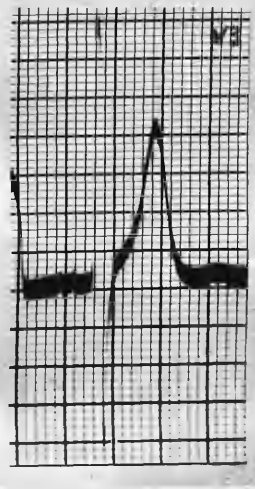
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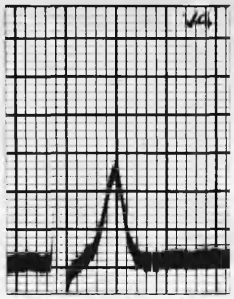
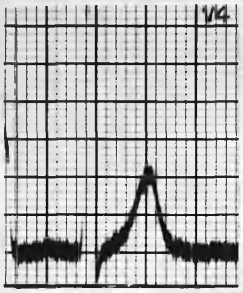
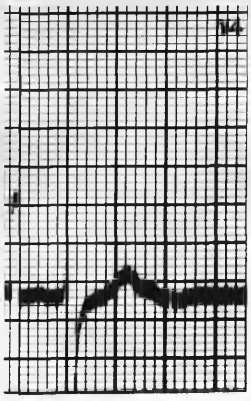
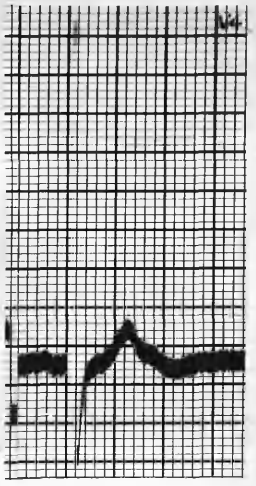
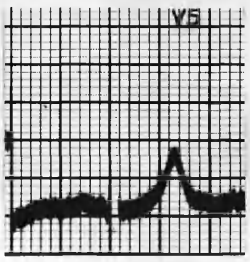
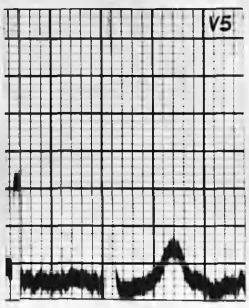


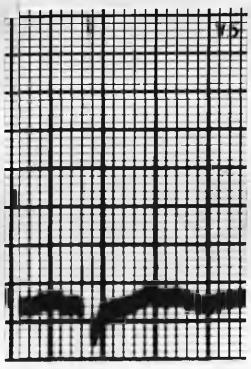


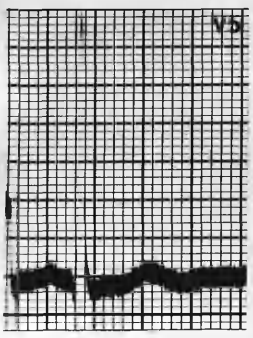


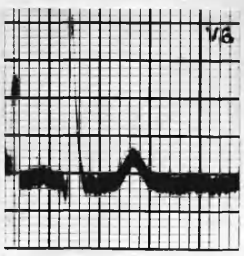


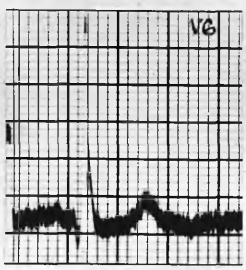


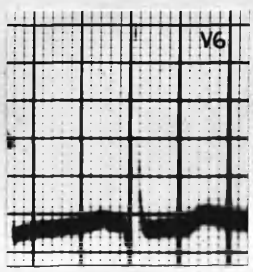


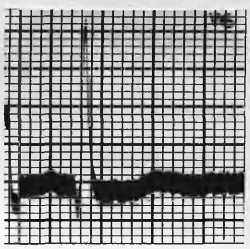


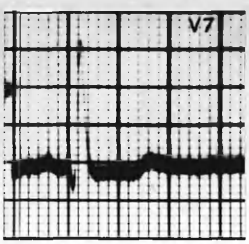


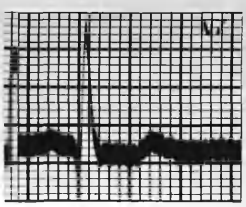








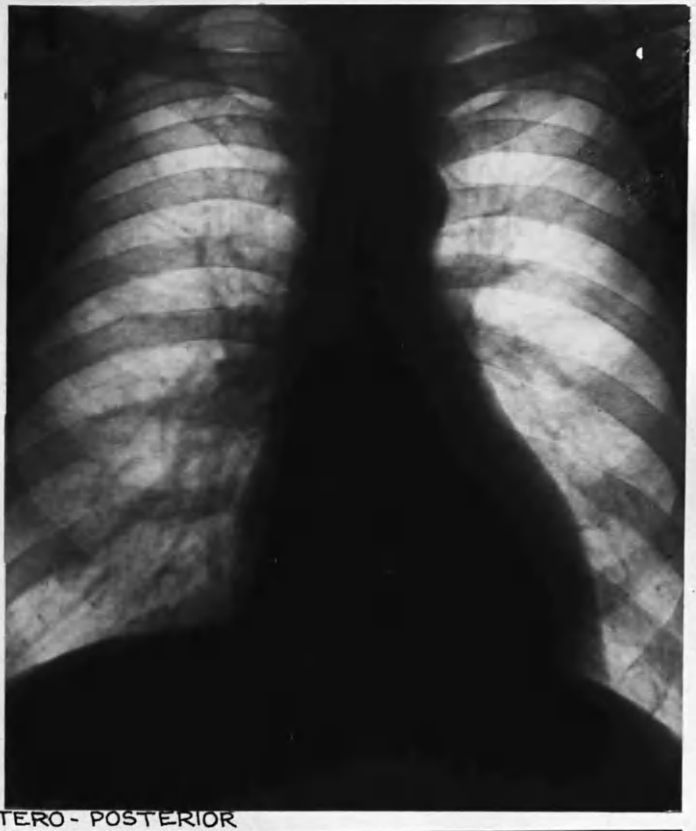




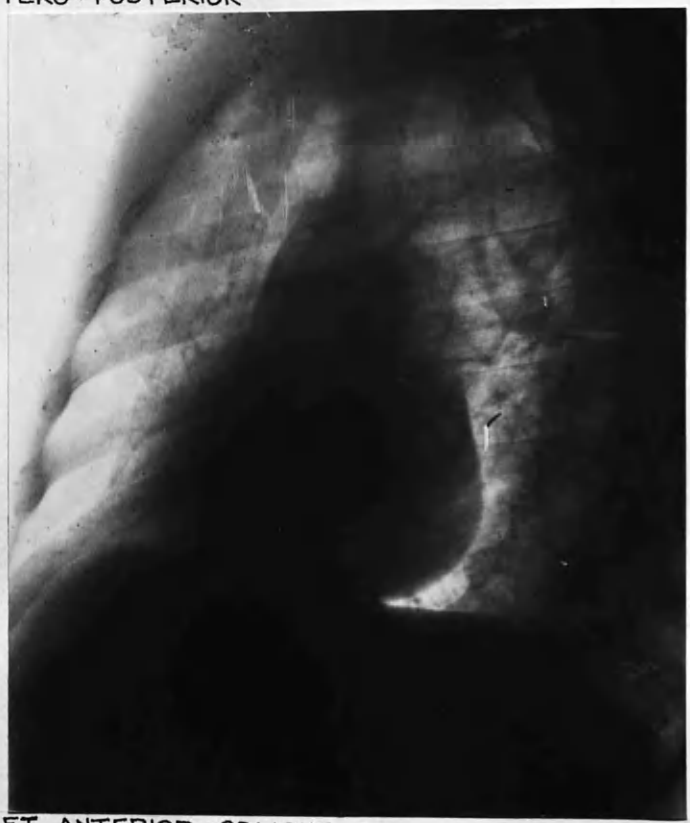
CASE 28,

J.A. MALE, 52 YEARS.

$2\frac{1}{2}$ MONTHS AFTER ONSET.	4 MONTHS AFTER ONSET.	$1\frac{1}{2}$ YEARS AFTER ONSET.	$1\frac{3}{4}$ YEARS AFTER ONSET.



ANTERO - POSTERIOR



LEFT ANTERIOR OBLIQUE

Case 29. P.F., male, 53 years.

This patient was admitted to a surgical ward as an abdominal emergency. The diagnosis of an acute intra-abdominal lesion was not sustained and no laparotomy was performed. Ten days after admission he was transferred to a medical ward. The first 12-lead electrocardiogram was obtained on the thirteenth day of illness. By that time his clinical condition was fair. The cardiac sounds were soft; B.P. was variable (160/70, 130/88, 100/62). Radiological examination showed a normal cardiac shadow. Previous to this illness, patient had suffered from exertional dyspnoea and intermittent claudication for nine years. These symptoms had followed an acute attack of retrosternal pain accompanied by collapse while walking but details of this illness were not available.

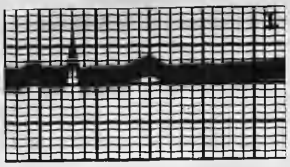
The electrocardiograms taken on the thirteenth day of the present illness show signs in lead VF diagnostic of posterior infarction; the QR complex measuring more than 5 millivolts shows a Q wave almost equal in amplitude to the succeeding R wave and occupying .03 sec. from onset to nadir. This pattern is carried into leads II and III. There is very slight bowing of the ST segment and T is of shallow diphasic type in leads VF and II.

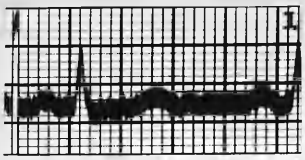
These ST and T signs alone constitute the evidence that infarction took place as recently as thirteen days before; otherwise the pattern may well be fixed from posterior infarction of nine years previously. The praecordial leads would favour the second possibility for there are neither high peaked T waves nor shortened S waves which are very common in the early weeks after infarction. The praecordial leads are within normal limits and are of a form commonly associated with healed posterior infarction. The clinical diagnosis of recent infarction is also open to doubt.

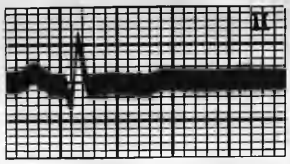
The patient finally left hospital free of symptoms. He was not seen till a year and a half later. He had resumed modified activity in his business and had only occasional pain in the chest on walking. B.P. was 110/60 and the cardiac sounds were of medium intensity and pure. An electrocardiogram shows the typical fixed pattern of posterior infarction. Lead VL now shows a relatively prominent R and small S waves compared to the equiphasic RS of the previous electrocardiogram, indicating that the electrical axis of the heart has shifted from vertical or, more probably, semi-vertical to intermediate.

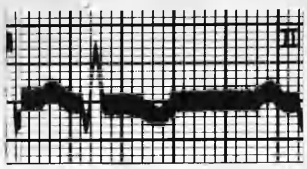
The apical and left ventricular leads now show high R waves with sagging ST-T segments suggesting early

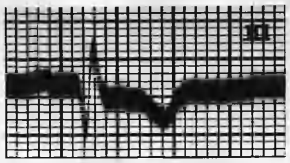
left ventricular hypertrophy. The delay in attaining the peak of R in leads V6 and V7 would indicate some local conduction defect of minor degree. These left ventricular changes are reflected in the radiological report of the same date, viz. "slight enlargement of the left ventricle." The change in electrical axis is also attributable to this cause. This case thus illustrates two of the sequelae of posterior infarction; on the one hand there is slight left ventricular hypertrophy which is probably a compensatory phenomenon and on the other there is a minor conduction defect in the postero-lateral wall implying fibrotic foci interspersed in sound muscle.

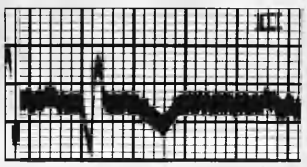


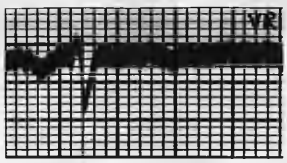


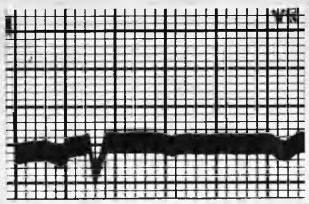


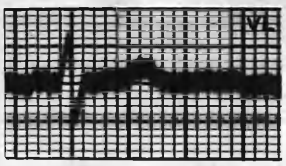


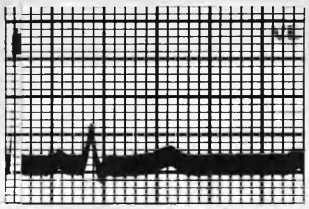


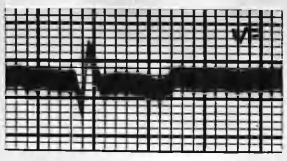


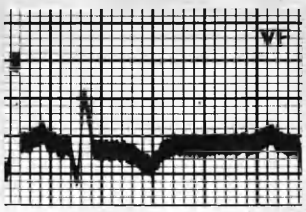






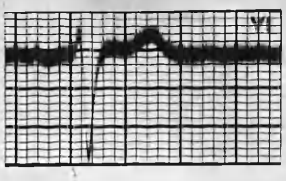


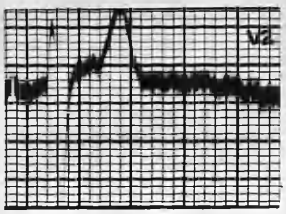


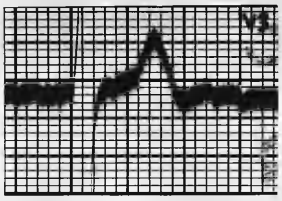


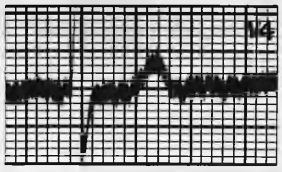
CASE 29,
P.F. MALE, 53 YEARS.
13 DAYS
AFTER ONSET.

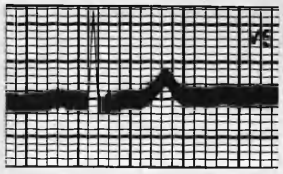
1½ YEARS
AFTER ONSET.

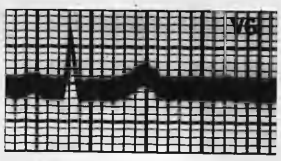


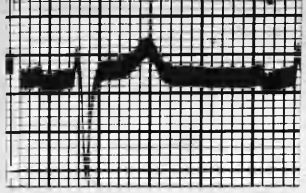


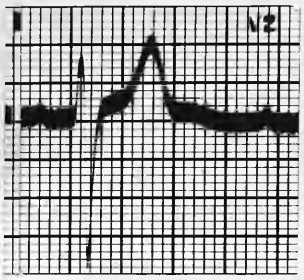


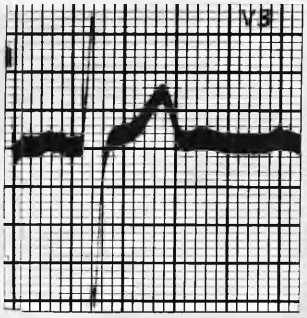


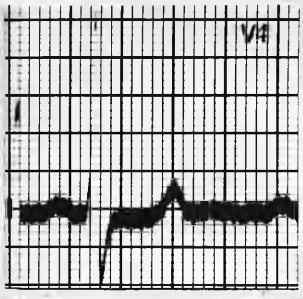


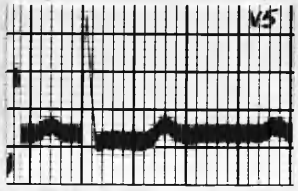


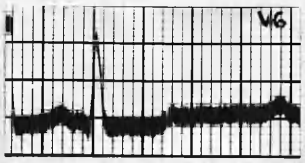


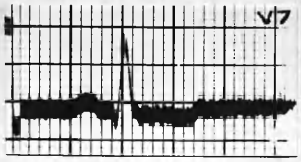






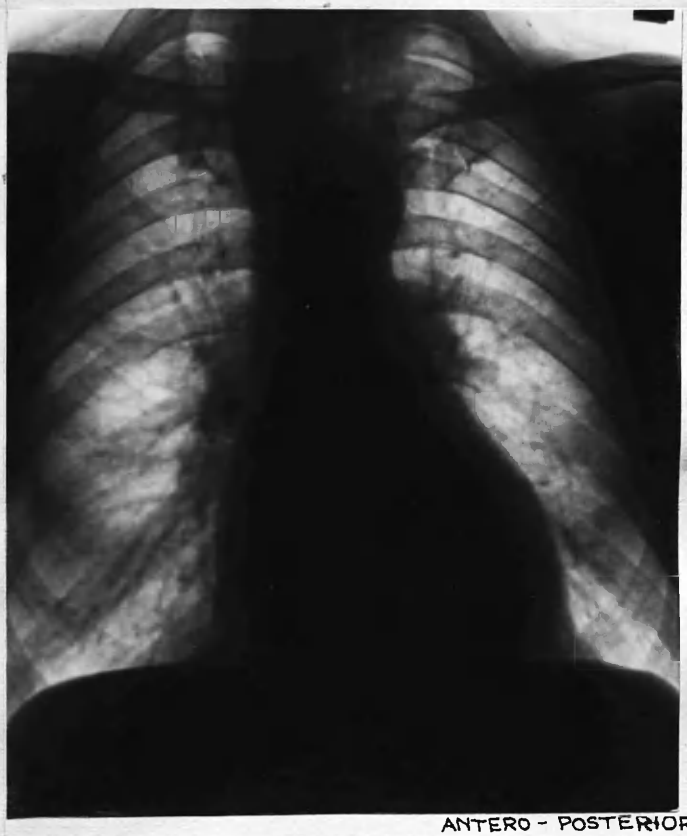




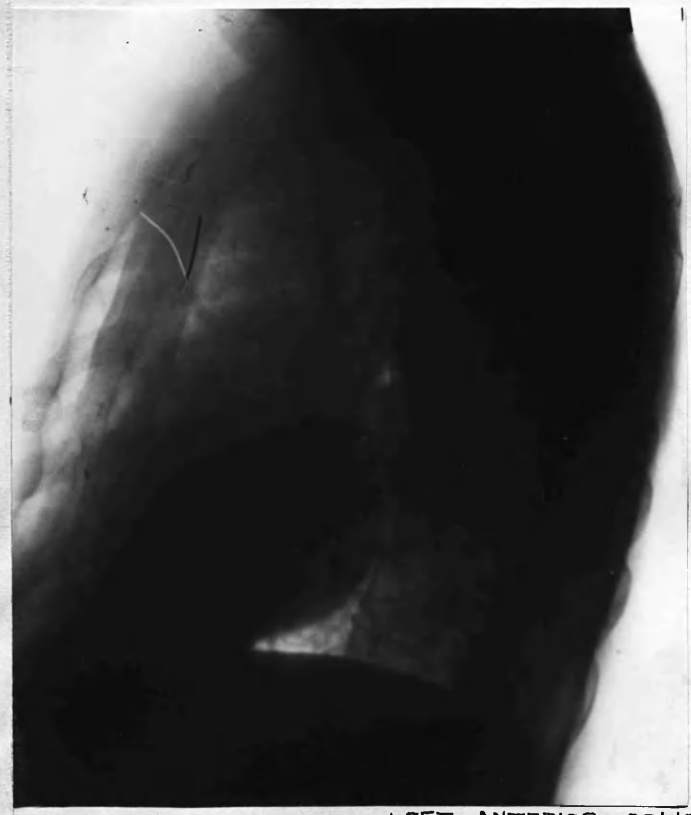


CASE 29,
P.F. MALE, 53 YEARS.
13 DAYS
AFTER ONSET.

1½ YEARS
AFTER ONSET.



ANTERO - POSTERIOR



LEFT ANTERIOR OBLIQUE

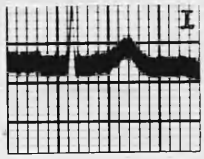
Case 30. W. G., male, 49 years.

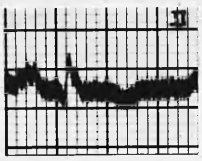
This man was seen at the out-patients' department two months after an attack of praecordial pain coming on at work and associated with some collapse. He remained in bed at home for six weeks. When seen as an out-patient, he felt well; B.P. was 130/75; the cardiac sounds were somewhat soft.

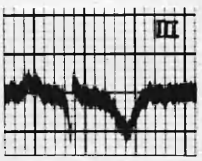
An electrocardiogram shows signs in leads VF, II and III very suggestive, if not diagnostic, of posterior infarct. Lead VL displays a prominent R wave suggesting that the electrical axis is intermediate, semi-horizontal or horizontal. The praecordial leads are within normal limits. Two years later the patient was seen again. He still had some discomfort in the chest on walking, and he had been unable to secure work light enough for him to undertake. The standard limb leads of an electrocardiogram taken at this time show little change. There is, however, slight widening of the QRS complex especially in lead II where it measures 0.1 sec. Similarly there is broadening of QRS in leads VR and VF. The latter displays a broad bifid Q wave occupying .04 sec. followed by a broad R wave. In addition the intrinsic deflection is

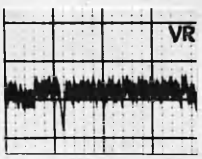
significantly later in lead VF than in praecordial leads over the left ventricle. It is concluded, therefore, that a conduction defect is present in the posterior wall of the left ventricle and is the result of the infarct. The praecordial leads show lengthening of S in leads V1 through V3 indicating increased negative potentials in the left ventricular cavity and heightening of R in leads V4 and V5. These signs are evidence of increased functional capacity in the undamaged regions of the wall. It is noteworthy that in this case the R wave in leads VL and VF shows very little increase in height, suggesting that the portions of left ventricular wall subtended by them have shared very little in any enhanced function probably because of the presence of scar tissue there or in the neighbourhood.

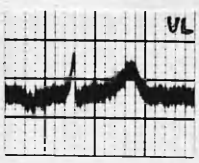
This case is an illustration of the late changes following a posterior infarct. While there is laminar or patchy fibrosis in a limited area of the posterior wall accounting for the fixed pattern of posterior infarct in the standard limb leads and causing a localised conduction defect, the praecordial leads indicate some compensatory enhancement of left ventricular function.

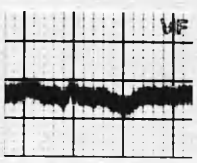




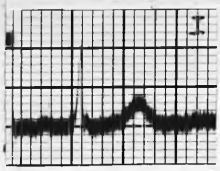


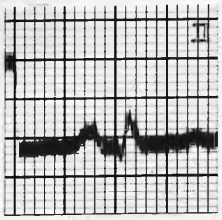


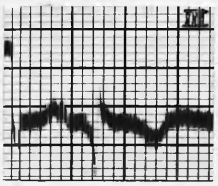


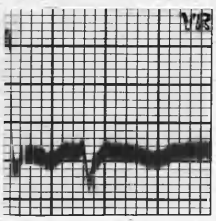


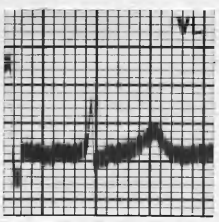
CASE 30,
W.G. MALE, 49 YEARS.
2 MONTHS
AFTER ONSET.

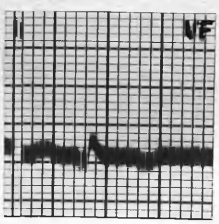




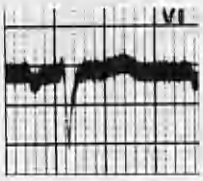


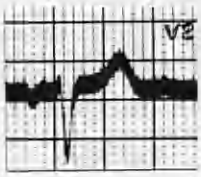


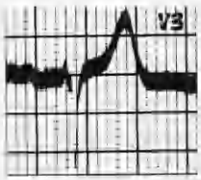


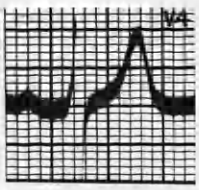


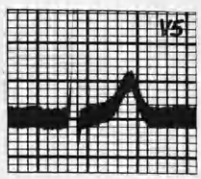
2 YRS 8 MTHS
AFTER ONSET.

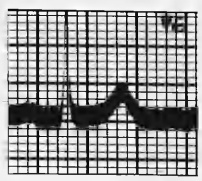




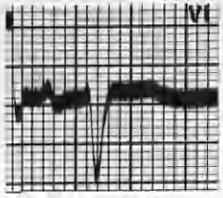


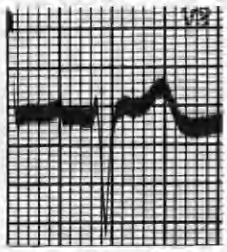


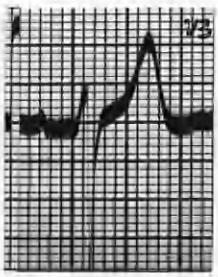


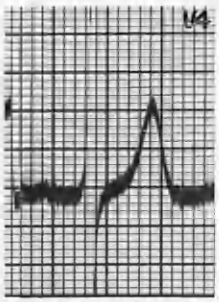


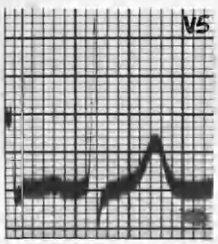
CASE 30,
W.G. MALE, 49 YEARS.
2 MONTHS
AFTER ONSET.

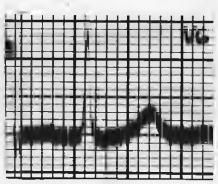


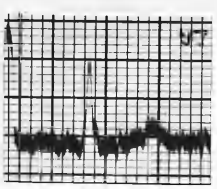












2 YRS 8 MTHS
AFTER ONSET.

Case 31. J.H., male, 57 years.

This patient, a steelworker of 57 years, had "a heart attack" two weeks before the first electrocardiogram was taken. He was seen at the out-patient department. Blood pressure was 88/60 and the cardiac sounds were of indifferent quality. Lead VF shows a Q wave of 3 mm. and a Q/R ratio of 60%; there is slight upward bowing of the ST segment and T is of shallow inverted type. These features indicate posterior infarction and are carried into leads II and III. The anterior praecordial leads show prominent R waves in leads V2 and V3 and large T waves in leads V1, V2 and V3 in which leads also the S waves are abnormally short. This configuration raises the possibility that the original pattern of these leads has been derived more from the left than from the right ventricle by counterclockwise rotation of the heart. Leads V2 and V3 are similar to lead VL which also derives its potentials from the uninfarcted free wall of the left ventricle. Nevertheless, the symmetrical shape of the large T waves in lead V1 through V3 is suggestive of the reciprocal influence of a posterior infarction, at least of its ischaemic zone which however would not be sufficient to heighten the R waves or truncate the S waves of leads V1, V2 and V3.

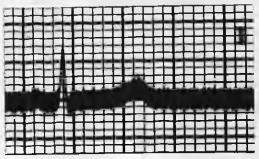
When seen again eight months later, the patient looked and felt well. He seldom had any chest pain and was only /

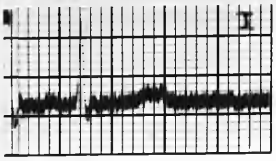
/ only slightly breathless on hills. Blood pressure was 150/80 and cardiac examination was negative. Radiologically, "the transverse diameter of the heart was within normal limits and pulsation appeared normal". An electrocardiogram now shows elongation of the Q wave in lead VF, the Q/R ratio being now over unity, the duration of the Q wave has increased to 0.03 sec. Since the infarct is now of some age, ST and T changes are of less significance. The increase in the size of the Q wave is reflected in leads II and III. The praecordial leads show some lengthening of the S waves in the anterior leads and some increase in the R waves in leads V2 to V5 inclusive.

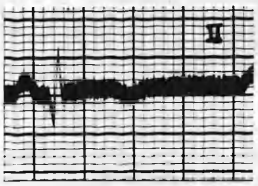
In explanation it may be argued that the heart may now be less counterclockwise rotated than previously and thus the anterior praecordial leads will have the more usual RS form. However there is no confirmation of change of axis in other leads. It is more probable that the new prominence of the S waves of leads V1, V2 and V3 represents some restoration of "cavity" potentials and is an indication of functional recovery which is also indicated by the slight increase in height of the R waves in the apical and adjoining leads - an increase which passes as far forwards as lead V2 if not V1 because of the counterclockwise rotation of the heart, causing the left ventricle to be accessible to the exploring electrode even in these anterior positions. The unchanging height of the R wave of lead VL /

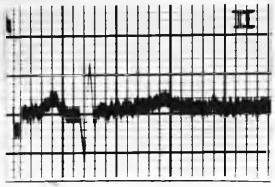
/ lead VL shows that any enhancement of left ventricular function has not been sufficient to affect the muscle subtended by the left arm electrode.

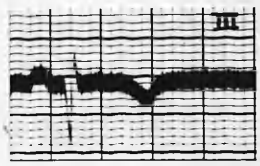
This case is an example of posterior infarction with excellent clinical recovery. Although foci of scar tissue remain in the posterior wall, there is adequate compensation electrocardiographically.

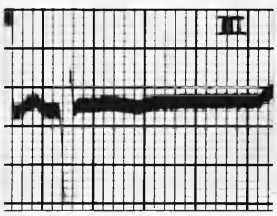


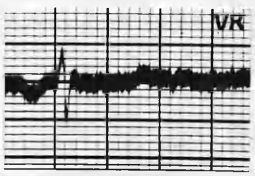


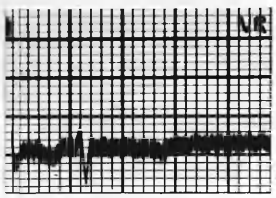


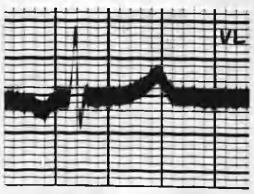


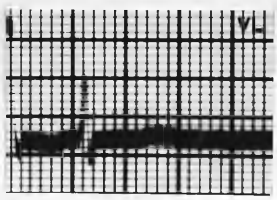


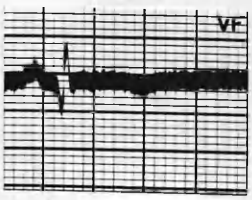


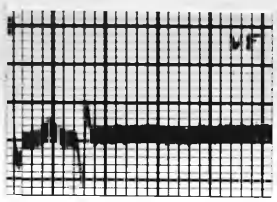








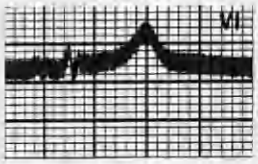


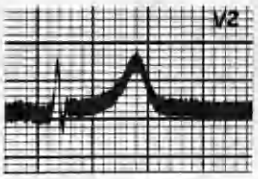


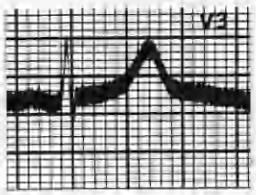
CASE 31, J.H. MALE 57, YEARS.

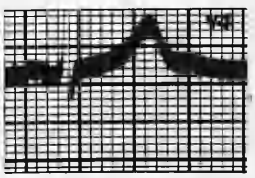
ABOUT 2 WKS AFTER ONSET.

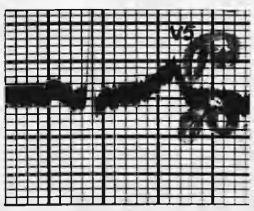
8 MONTHS AFTER ONSET.

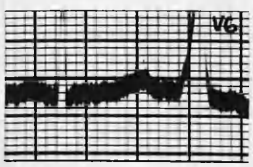


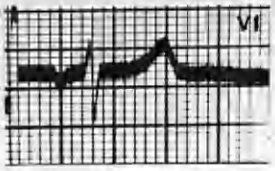


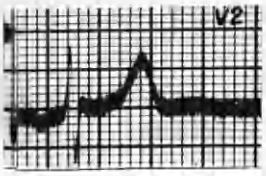


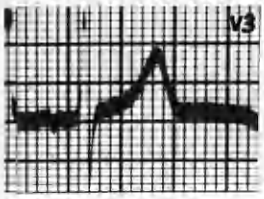


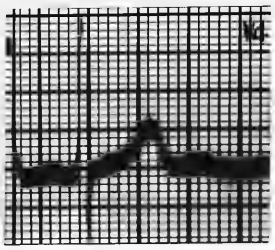


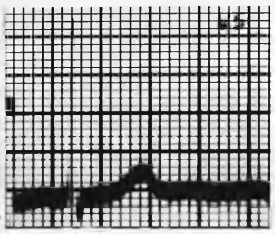


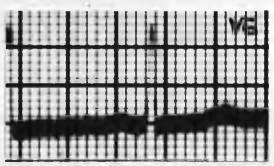


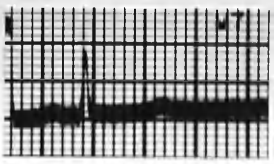








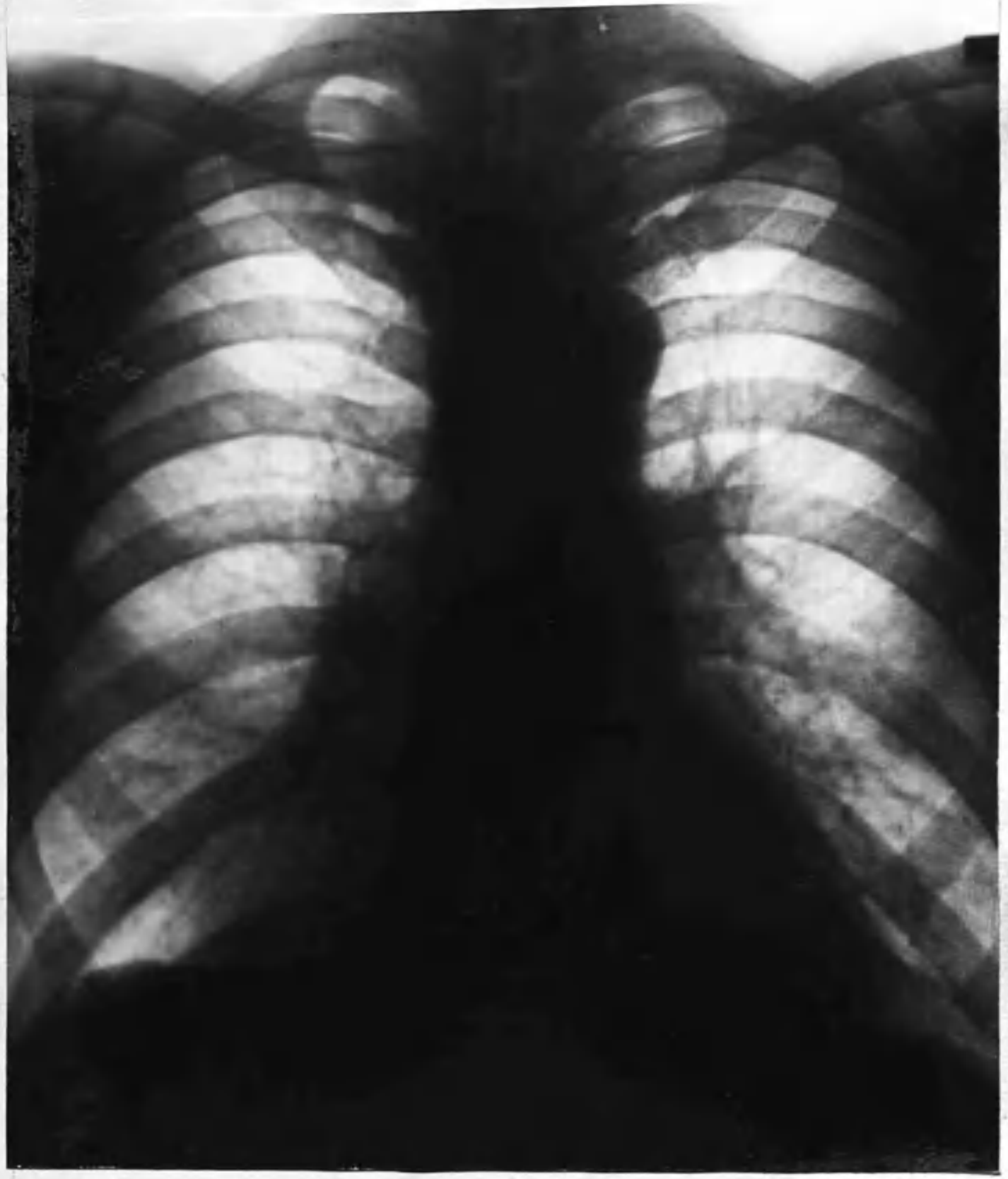




CASE 31, J.H. MALE, 57 YEARS.

ABOUT 2 WKS AFTER ONSET.

8 MONTHS AFTER ONSET.



ANTERO - POSTERIOR

Case 32. J.S. Male. 61 years.

This patient had an attack of constrictive retrosternal pain which confined him to bed at home for several months. His first attendance at the out-patient department was not till five months after onset. Blood pressure was 204/110. There was slight increase of cardiac dullness and the cardiac sounds were soft. An electrocardiogram taken at that time shows a broad splintered QS complex in lead VF with sharply inverted T wave. If the heart is horizontal such a QS complex may indicate a septal infarction, but if it is intermediate (or vertical) it may be a manifestation of a practically complete transmural posterior infraction. The prominent R wave of lead VL does not differentiate the horizontal from the intermediate position in both of which it occurs. The praecordial leads show only a slight reciprocal increase in the R and T waves in leads over the right side of the praecordium with S waves of normal length. Lead V6 shows a sudden fall in height of the T wave with very slight bowing of the ST segment. These features would suggest that the electrode in this position was approaching the neighbourhood of the infarcted area and, if so, the probability is that the

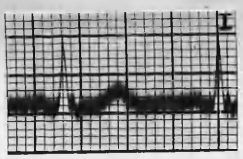
original infarct was in the posterior free wall of the left ventricle with some lateral extension rather than in the septum.

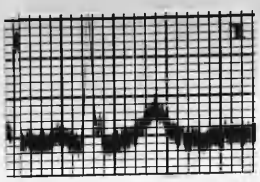
The patient made a good recovery and when seen more than one year later he admitted only slight discomfort in the left chest on exertion. Blood pressure was 160/90. There was slight cyanosis and the cardiac sounds were rather soft.

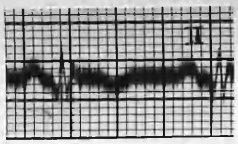
An electrocardiogram now shows the fixed pattern of a posterior infarction in the standard limb leads with increase in the R wave in leads I and II and the S wave in lead III. A similar increase in the R wave occurs in lead VF indicating the resurgence of some muscle elements within the infarcted area. Lead VL shows a striking increase in the R wave which is a reflection of its increased height in all the praecordial leads chiefly V3, V4 and V5. Simultaneously there is an increased depth of S in the anterior praecordial leads chiefly V2 and V3. This pattern is very suggestive of considerable scar tissue at the site of infarction. Leads V6 and V7 show further changes; the QRS complex has increased to .12 sec. in duration and there is conspicuous notching of the descending limb of the R wave.

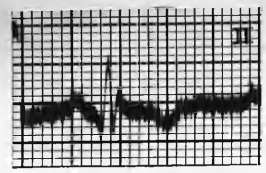
This new feature indicates a defect of conduction in the lateral wall of the left ventricle. The sagging of the ST segment and the diphasic T wave of these leads are secondary to the conduction defect. These facts are in accord with some encroachment of the infarct on to the lateral wall. Radiologically there was slight enlargement of the left ventricle but no abnormal pulsation was seen.

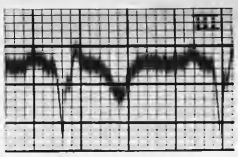
This case is an example of a healed infarction of the posterior or posterolateral wall with compensatory hypertrophy of left ventricle, scarring at the site of infarction and a residual conduction defect in the lateral wall.

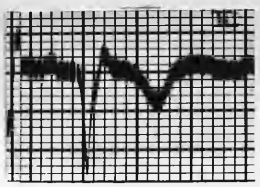


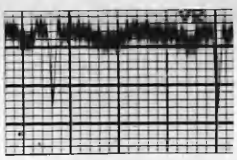


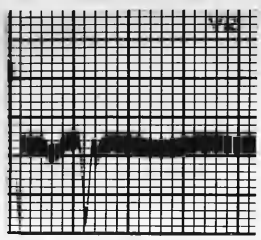


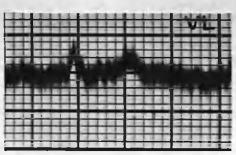


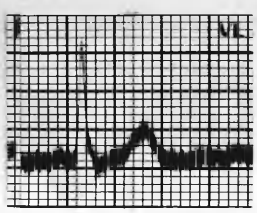


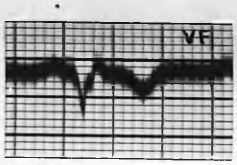


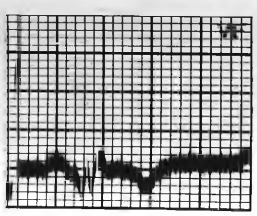








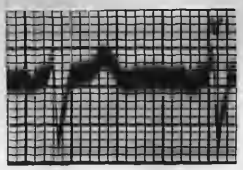


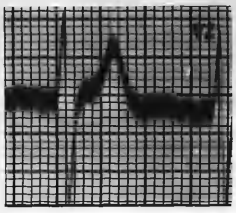


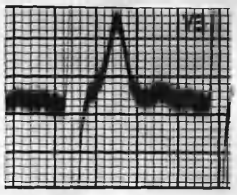
CASE 32,
J.S. MALE, 61 YEARS.

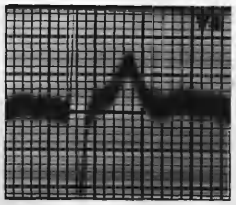
5 MONTHS AFTER ONSET.

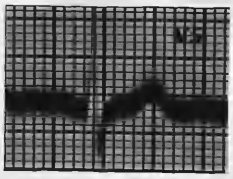
17 MONTHS AFTER ONSET.

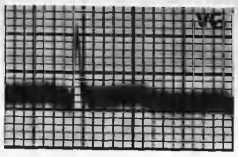




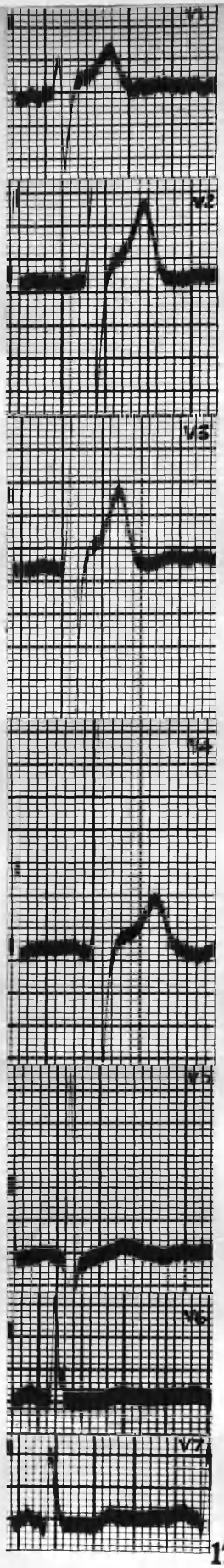




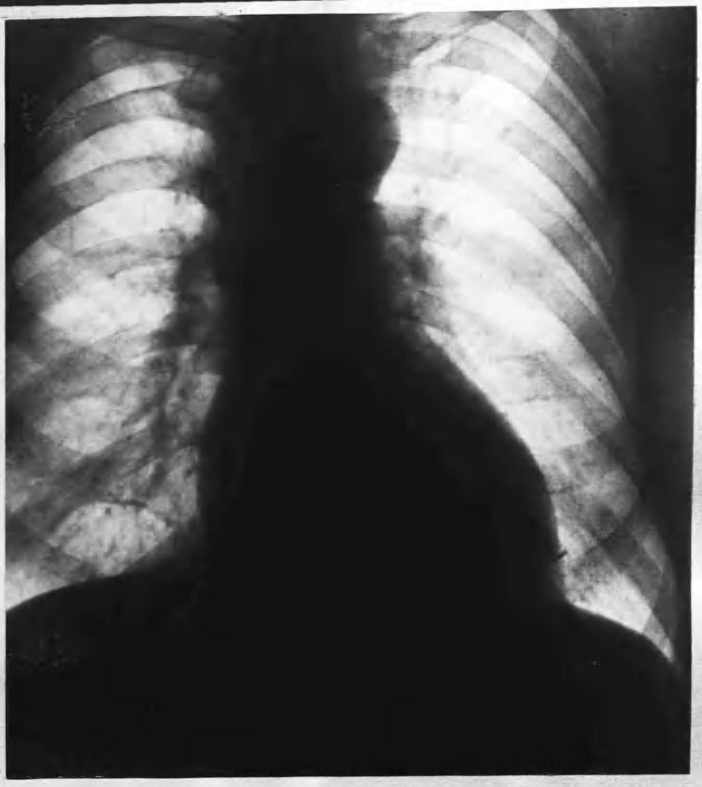




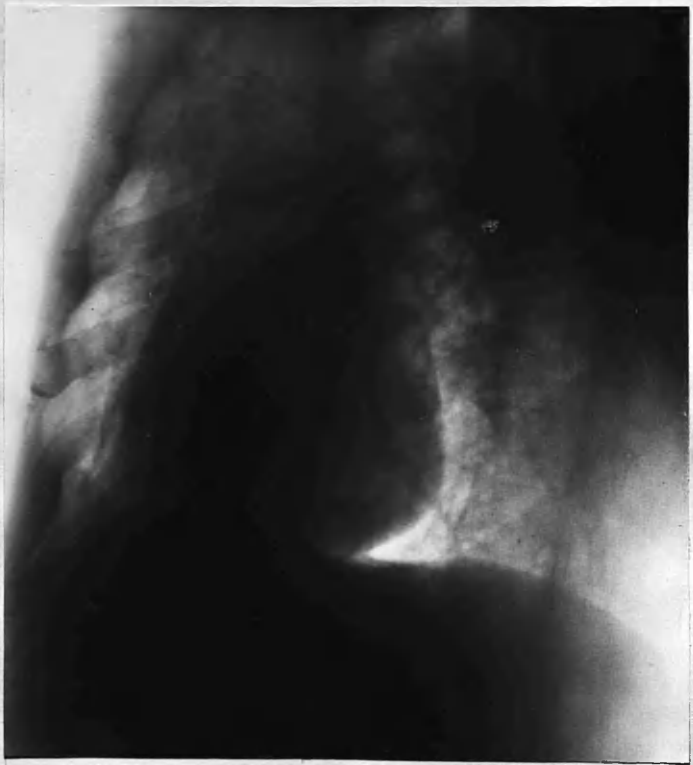
CASE 32,
J.S. MALE, 61 YEARS.
5 MONTHS
AFTER ONSET.



17 MONTHS
AFTER ONSET.



ANTERO - POSTERIOR



LEFT ANTERIOR OBLIQUE

Case 33. A.M., male. 57 years.

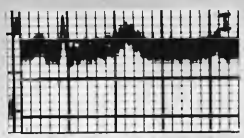
This patient gave a history of prolonged retrosternal pain unaccompanied by collapse or by a fall in blood pressure. Although the diagnosis of myocardial infarction was made, the family practitioner had not considered hospital admission necessary. It was not till two and a half weeks after onset that an electrocardiogram was obtained. It shows signs diagnostic of posterior infarction in leads VF, II and III. It is clear that the electrical potentials of the left leg are relatively small and have influenced leads II and III less than those of the right arm and left arm respectively; hence lead II is more or less the reciprocal of lead VR and lead III of lead VL - a relationship which is maintained throughout the series. Lead VL resembles leads V1 and V2 indicating that the left arm has derived its potentials from the epicardial surface of the right ventricle, a relationship which implies some clockwise rotation of the heart around its long axis such as commonly occurs in a vertical heart. The same considerations may explain the RS patterns in the praecordial leads, but a minor degree of right ventricular hypertrophy cannot be excluded especially in view of the mild

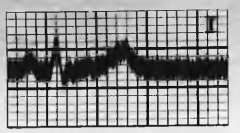
emphysema noted clinically and radiologically. In either case the RS pattern would appear to be but little influenced by the presence of the posterior infarction: R is not unduly tall nor is S unduly short and its form at six weeks is practically the same as at two and a half weeks. The T waves, however, show typical changes - tall and spike-like - in leads over the right side of the praecordium, indicating a reciprocal effect from a zone of ischaemia rather than from the infarct itself. In the early weeks only, the T waves are sharply inverted in the axillary leads indicating the direct effect of a temporary zone of ischaemia surrounding the infarct in this region. The ST segment in leads over the right side of the praecordium rises to its normal position above the base-line in the tracings at six weeks.

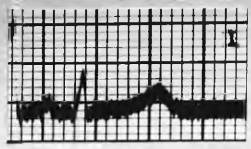
This patient made an excellent recovery and returned to work as a railway guard. He was seen again almost two years later. The electrocardiogram now shows the signs of a well healed posterior infarct.

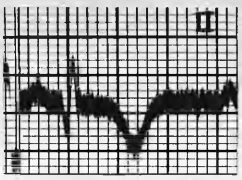
Striking changes are now seen in lead VR (and its reciprocal, lead II). In lead VR, S is now a deep deflection, indicating a return of the full negative potentials of the left ventricular cavity. The same

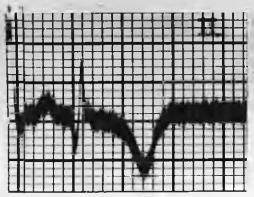
elongation of S is seen in leads VI through V4. For the same reason there is a slight increase in height of R in leads V4 and V5, but it rapidly decreases in leads V6 and V7 because of the vertical position of the heart, the broad shape of the chest, the interposition of emphysematous lung and the approach of the electrode, in the V7 position, to the region of the scar tissue in the ventricular wall. Over the right side of the praecordium, the wave R remains very constant in size. Its failure to decrease with healing of the infarct favours the view that its original slight prominence was due to early right ventricular hypertrophy and not to the diminution of forces normally antagonistic to R in these leads, caused by the infarct.

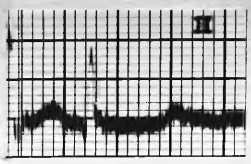


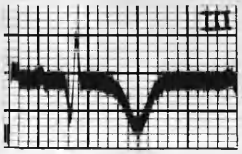


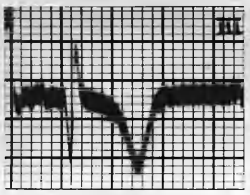


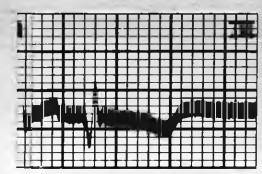


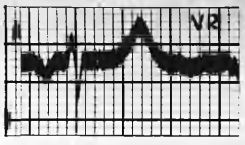


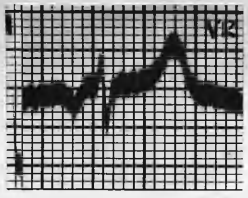


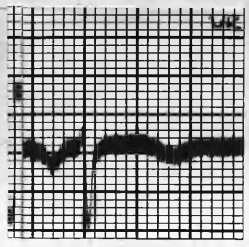


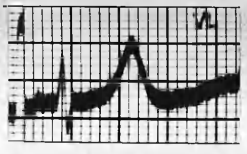


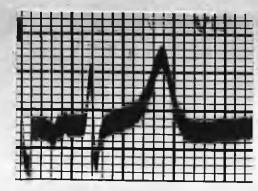


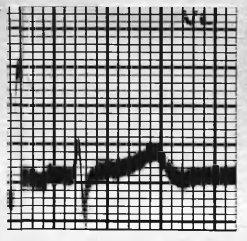


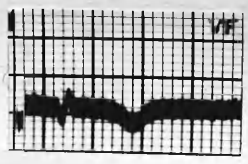


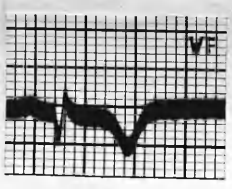


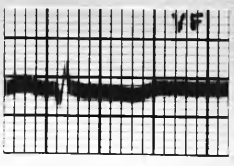










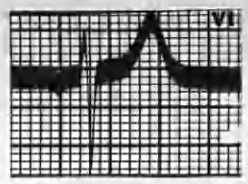


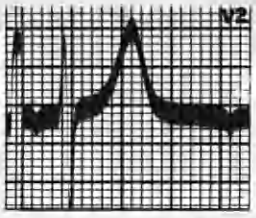
CASE 33,
A.M. MALE, 57 YEARS.

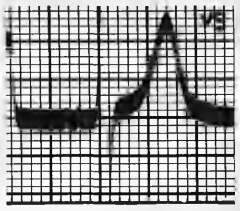
2½ WEEKS AFTER ONSET.

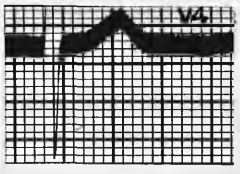
6 WEEKS AFTER ONSET.

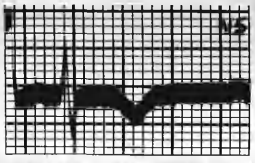
22 MONTHS AFTER ONSET.

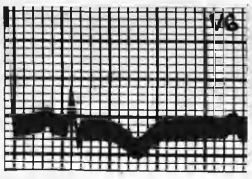




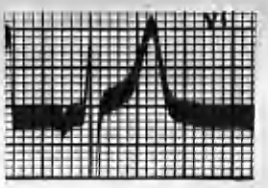


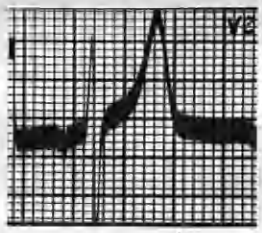


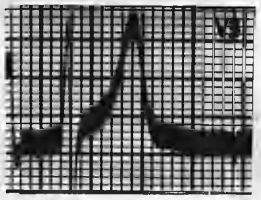


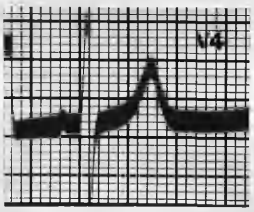


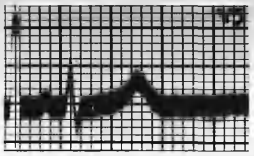
CASE 33,
A.M. MALE, 57 YEARS.
2½ WEEKS
AFTER ONSET.

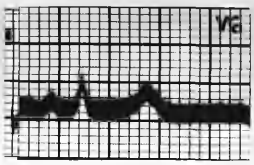




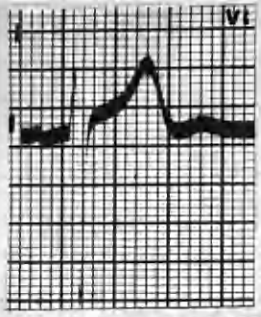


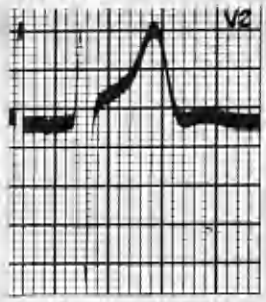


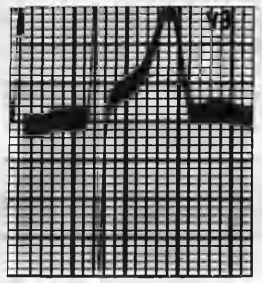


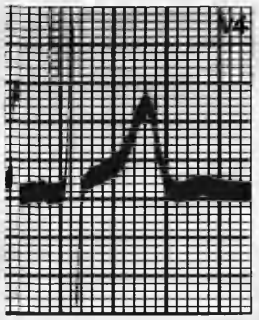


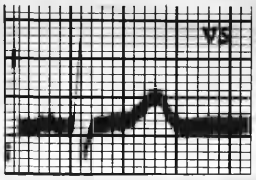
6 WEEKS
AFTER ONSET.

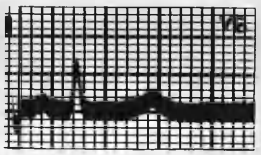


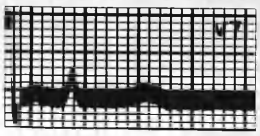




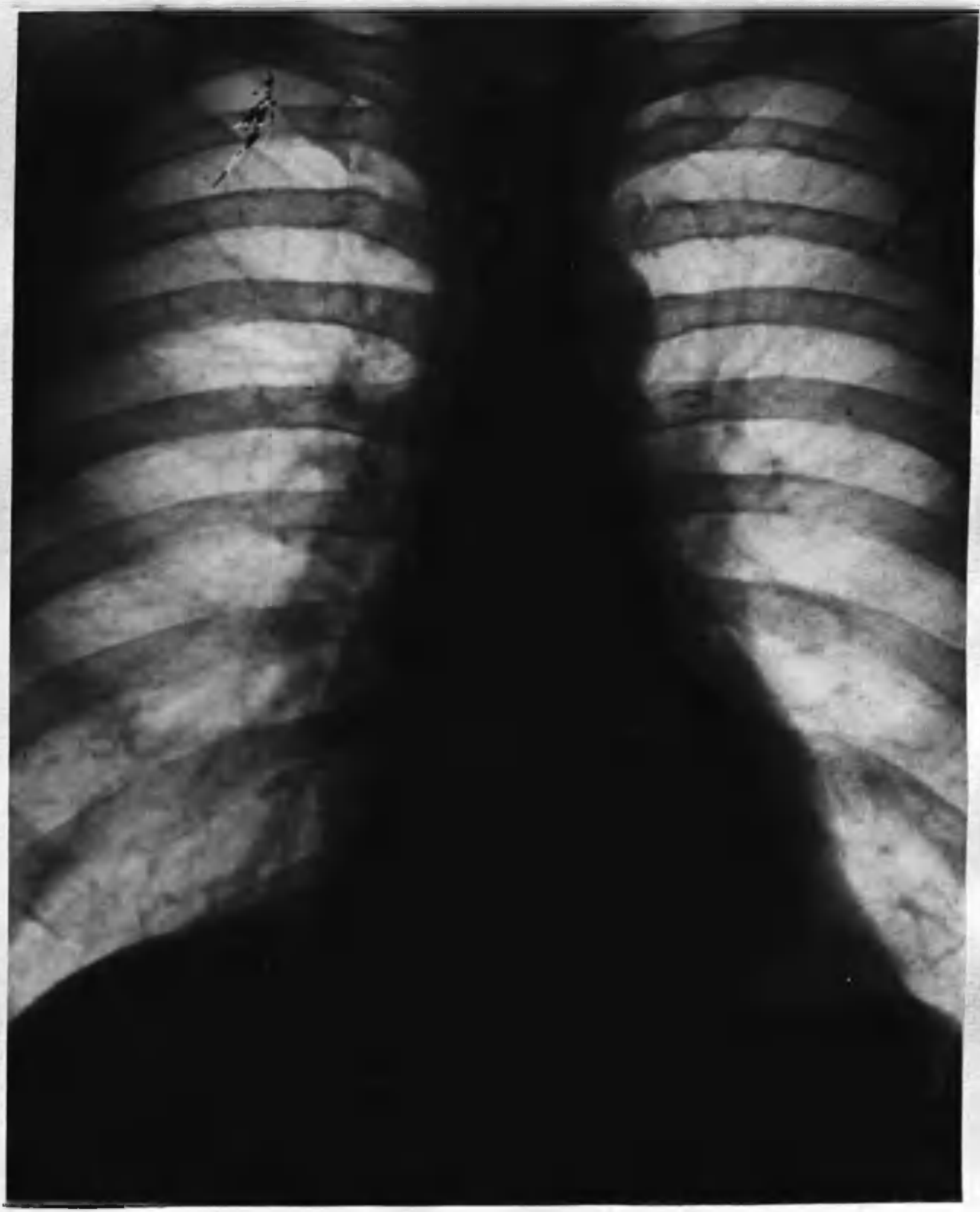








22 MONTHS
AFTER ONSET.



ANTERO - POSTERIOR

Case 34. S.Y., male, 56 years.

This patient conducted an active medical practice in spite of anginoid pain on exertion recurring for a year prior to the attack of pain which brought him into hospital. This attack occurred while at rest, was associated with collapse and required morphine for its relief. Only standard limb leads were obtained in the first instance nine hours after onset, and it was not till one and a half days after onset that a full thirteen-lead electrocardio-gram was obtained. The evolution of lead III in this short interval would leave little doubt as to the diagnosis of posterior infarction. In addition lead VF displays slight elevation of ST but no Q wave in the small broad ventricular deflection. In spite of these significant changes in the standard and unipolar limb leads, the praecordial leads maintain a normal appearance except for flattening of T in the axillary leads.

The patient remained sharply ill during his first week in hospital with widely radiating anginoid pain recurring four or five times daily and blood pressure remaining at subnormal levels. An electrocardiogram taken two and a half days after onset shows a rapid evolution of the ST-T segment in lead VF in a fashion suggestive of pericarditis, but it is not till four and a half days after onset that classical criteria for the diagnosis of posterior infarction are seen in this lead,/

/lead, viz. the QR complex, increased to 0.5 millivolt, shows a Q wave measuring 0.04 sec. from onset to nadir and having an amplitude of more than 25% of the succeeding R wave. The most probable explanation for the delay in these new signs is that the infarction has only now spread subendocardially to the diaphragmatic wall and thus influenced the pattern of lead VF or that the infarction previously patchy or laminar has now become transmural if it can be assumed, in view of the absence of the Q wave in the first two tracings, that the islets of infarcted muscle did not originally interfere with the conducting and contracting power of the ventricular muscle during the first two and a half days of illness. In either case R should have decreased, not increased,in height although the retention of a considerable R wave in lead VF is quite compatible with a transmural infarction, since the area subtended by lead VF is large enough to include undamaged muscle. It is unlikely that an axial shift to bring the original infarction more directly in opposition towards the left leg, alone accounts for the appearance of the Q wave in lead III because the RS deflections of the praecordial leads are not affected. Hence the likeliest explanation is spread of infarction in the diaphragmatic wall.

At this stage, viz. four and a half days after onset a very small R wall has returned to lead VR and the R wave of lead VL previously truncated is now becoming taller /

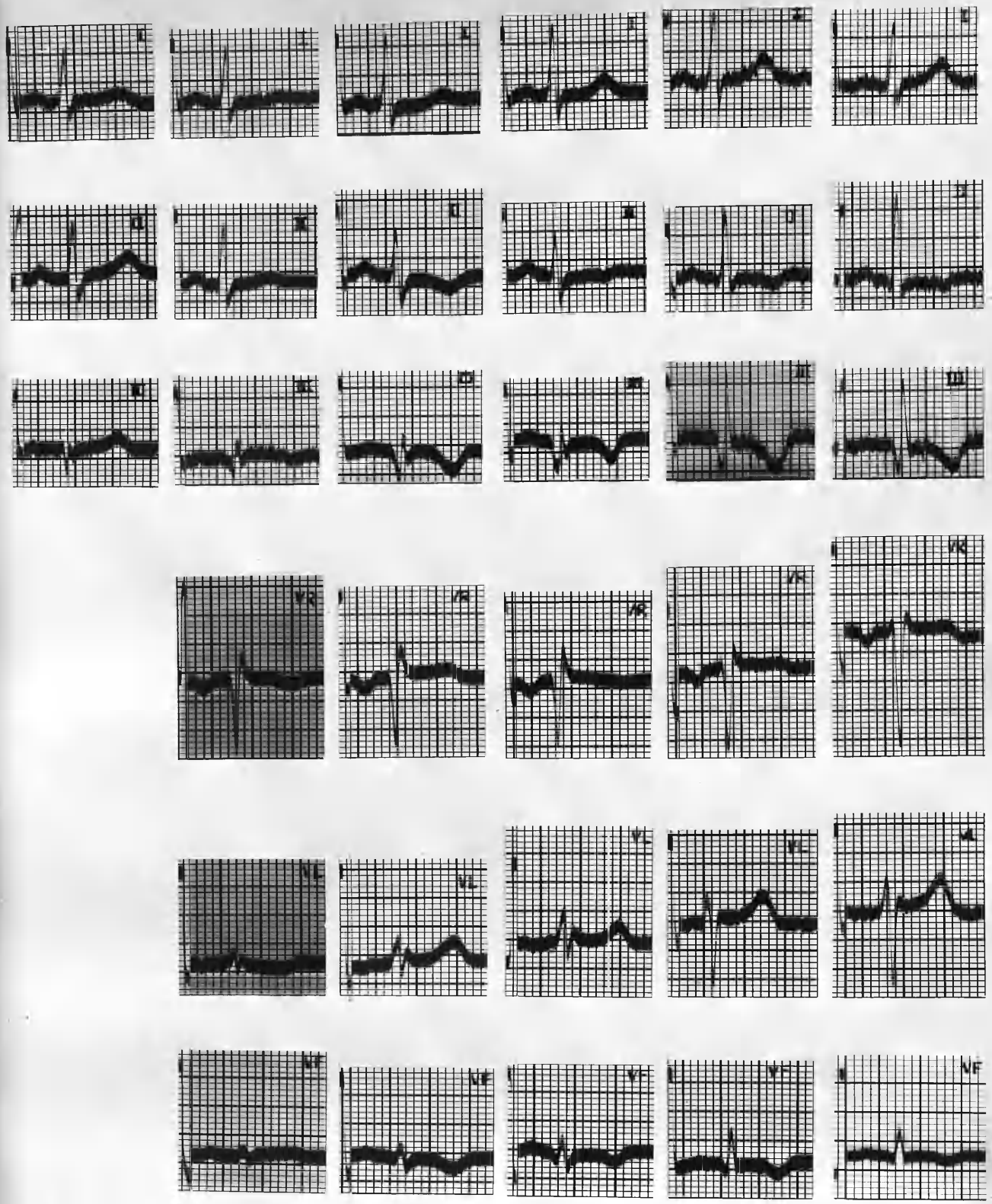
/taller and sharper. In view of the vertical position of the heart, as established subsequently, both leads VR and VL face the right side of the septum as well as the ventricular cavity. The original absence or diminution of the R wave has been due to involvement of the left side of the septum; local recovery is demonstrated by the signs noted above. Thus the original infarction has been posteroseptal in situation; at four and a half days after onset, there is spread in the diaphragmatic wall but some recovery in the septum.

Restorative changes in this case are well seen in the later electrocardiograms, at six and at eight weeks after onset. Lead VL shows a striking increase in S and the final pattern is similar to any lead facing the epicardial surface of the right ventricle. Thus the heart is in a vertical or semi-vertical position electrically.

In leads V1 through V4 and in lead VR, S has also increased considerably in depth. These changes are due to a restoration of the potentials of the left ventricular cavity, which are transmitted in normal manner through the septum and right ventricle, as these structures become depolarised, to the anterior praecordial leads which record them as a negative S wave. This return of left ventricular function has caused practically no increase of R in the apical lead or in the axillary leads for two reasons. A vertical or semi-vertical position of the heart tends to/

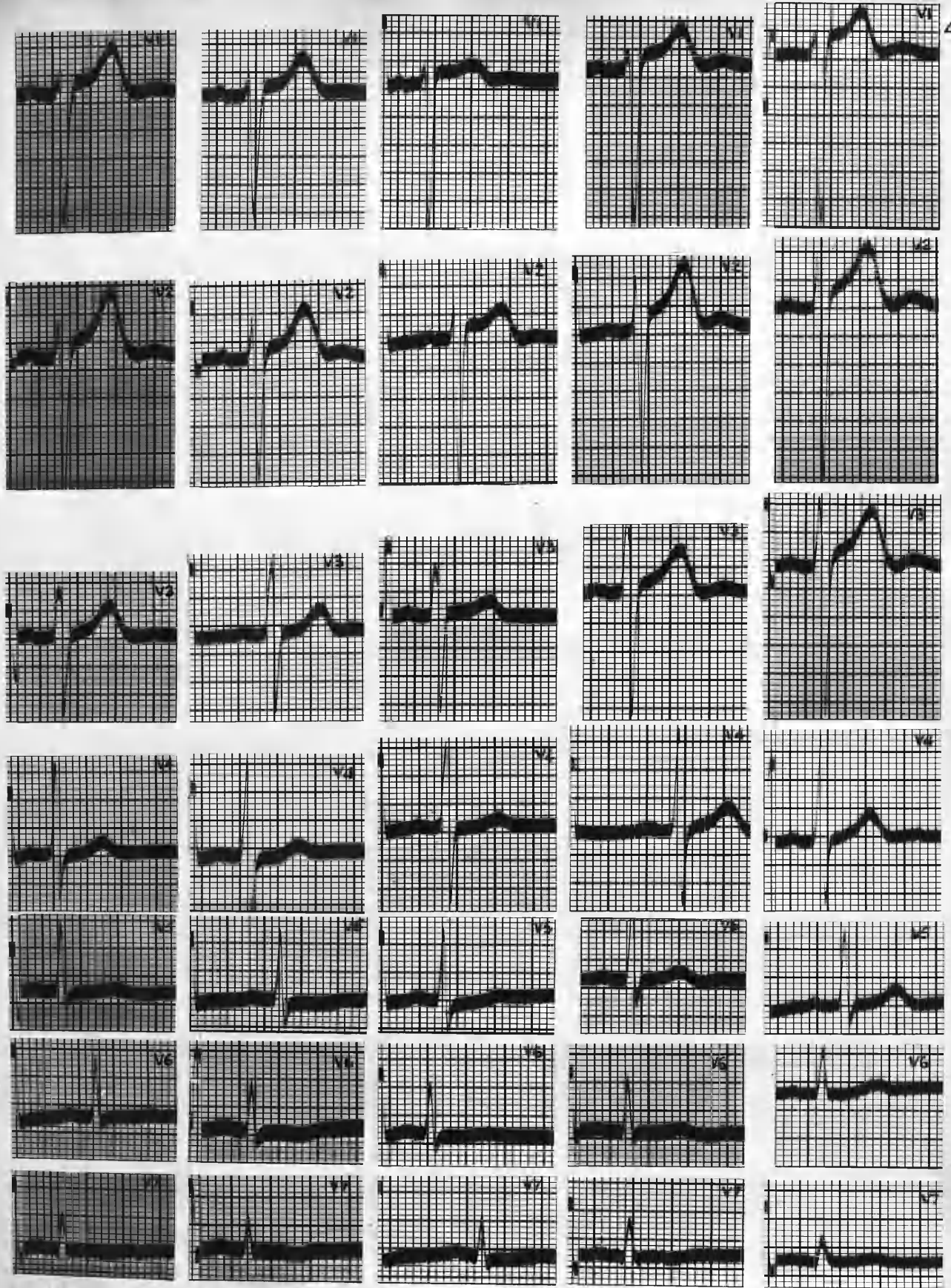
/to prejudice the recording of high R waves in leads V4 through V6 and in lead V7 the small broad R wave with slurred upstroke indicates a conducting lesion in the free wall of the left ventricle, a residue from some patchy involvement at the edge of the infarct. The restoration of left ventricular activity is expressed in the standard limb leads to the extent that the deflections in lead VF being small, lead III is practically the reciprocal of lead VL; hence the Q, tall R and large inverted T of lead III. Hence this Q3 is less the residue of posterior infarction than the result of initial positivity of the left arm.

This case presents the changes in a vertical or semi-vertical heart due to a posterior infarction, which is fairly well advanced at eight weeks in the healing process. By this time the patient was progressing satisfactorily and after a long convalescence, he was able to resume medical practice in a modified way.



CASE 34, S.Y. MALE, 56 YEARS.

9 HRS AFTER ONSET. | 1½ DAYS AFTER ONSET. | 2½ DAYS AFTER ONSET. | 4½ DAYS AFTER ONSET. | 6 WKS AFTER ONSET. | 8 WKS AFTER ONSET.



CASE 34, S.Y. MALE, 56 YEARS.

1½ DAYS AFTER ONSET.	2½ DAYS AFTER ONSET.	4½ DAYS AFTER ONSET.	6 WEEKS AFTER ONSET.	8 WEEKS AFTER ONSET.

Case 35. Miss M.M., 61 years.

This patient had had angina of exertion for three years. The pain radiated from the chest into the neck but not into the arms. For a year preceding hospital admission radiation to the jaw and into the legs occurred. A prolonged and widespread attack of pain was the immediate reason for admission to hospital. There was little peripheral vascular failure; the blood pressure fell to 90/60 on the third day of illness but thereafter was 120/80. Radiologically there was prominence of the left ventricle. Progress was uneventful and the patient left hospital after three months stay. She was free of pain but exercise tolerance was poor. For the subsequent two years during which information was obtainable her health was fair but dyspnoea was readily induced by exertion.

An electrocardiogram taken one week after onset of illness shows a splintered QS deflection in lead VF along with elevation of ST and diphasic T waves. In view of the tall R wave in lead VL the electrical position of the heart is either horizontal, semihorizontal or intermediate and, of the three possibilities, it is probably horizontal because of the prominence of the left

ventricle, noted radiologically. If so, the QS deflection in lead VF signifies septal involvement, the infarct being presumably posteroseptal in situation. The signs of acute infarction are carried into leads II and III. The praecordial leads show depression of the ST segments, but this is by no means a depression reciprocal to acute posterior infarction. In the first place it is confined to leads V4 to V7 inclusive and is not seen more anteriorly; the segment itself is bowed in an upward direction and it is associated in leads V5, V6 and V7 with diphasic T waves. These signs are very probably due to a combination of widespread subendocardial ischaemia and left ventricular hypertrophy. In the same electrocardiogram the arched ST - T formation in leads V1, V2 and V3 suggests some minor pericardial involvement. Alone of the unipolar leads, lead VL by virtue of its saddle-shaped ST - T form and the subsequent evolution of its T wave, shows the reciprocal effect of posterior infarction.

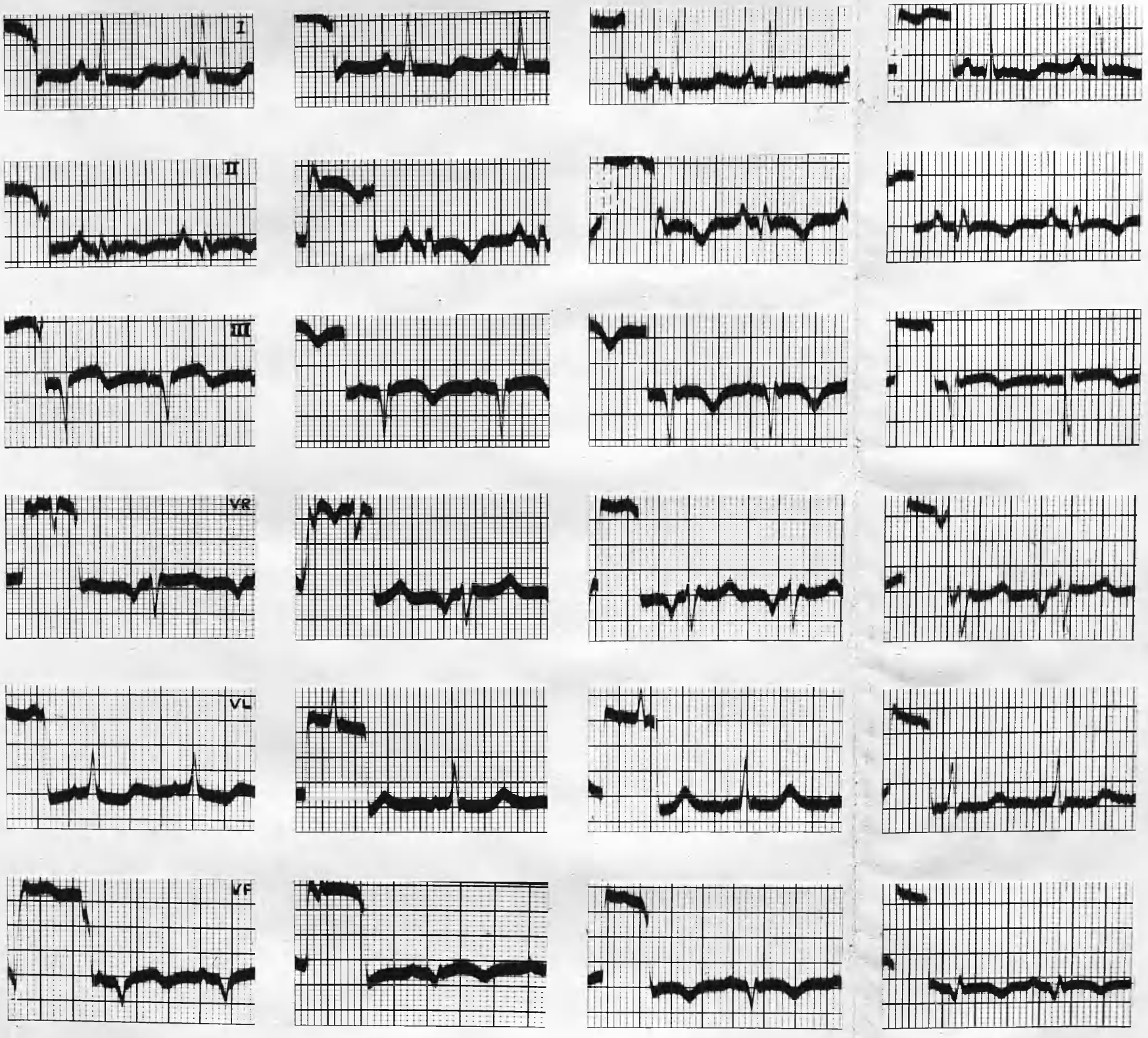
Two and a half weeks later a small R wave has returned to lead VF and the QS deflection is not so deep. The T waves of this lead and of leads V4 to V7 inclusive are now sharply inverted and the ST segments of the

latter four leads are now rising to the iso-electric level. There is therefore persistent widespread ischaemia of the lateral ventricular wall although it can no longer be localised to the subendocardium.

A further two weeks later expected sequential changes are seen in the standard and unipolar limb leads but there is little change in the praecordial leads. After a further interval of three weeks, i.e. eight and a half weeks after onset there is a little progress. Lead VL, deriving its potentials from the left ventricular wall, now shows an iso-electric ST segment. This is also seen in leads V5 to V7 and in standard limb lead I. The T waves of the apical and axillary leads remain inverted however.

This case illustrates the scantiness of reparative processes in some instances. The chief difference between the electrocardiograms at one week and at eight and a half weeks after onset is the diminution in the signs of subendocardial ischaemia of the lateral heart wall with reversion to signs which indicate either an ischaemic process in general or left ventricular hypertrophy or dilatation, probably old-standing. The return of the R wave to lead VF and the

S wave to lead VL are the only signs of some patchy recovery in the infarct.

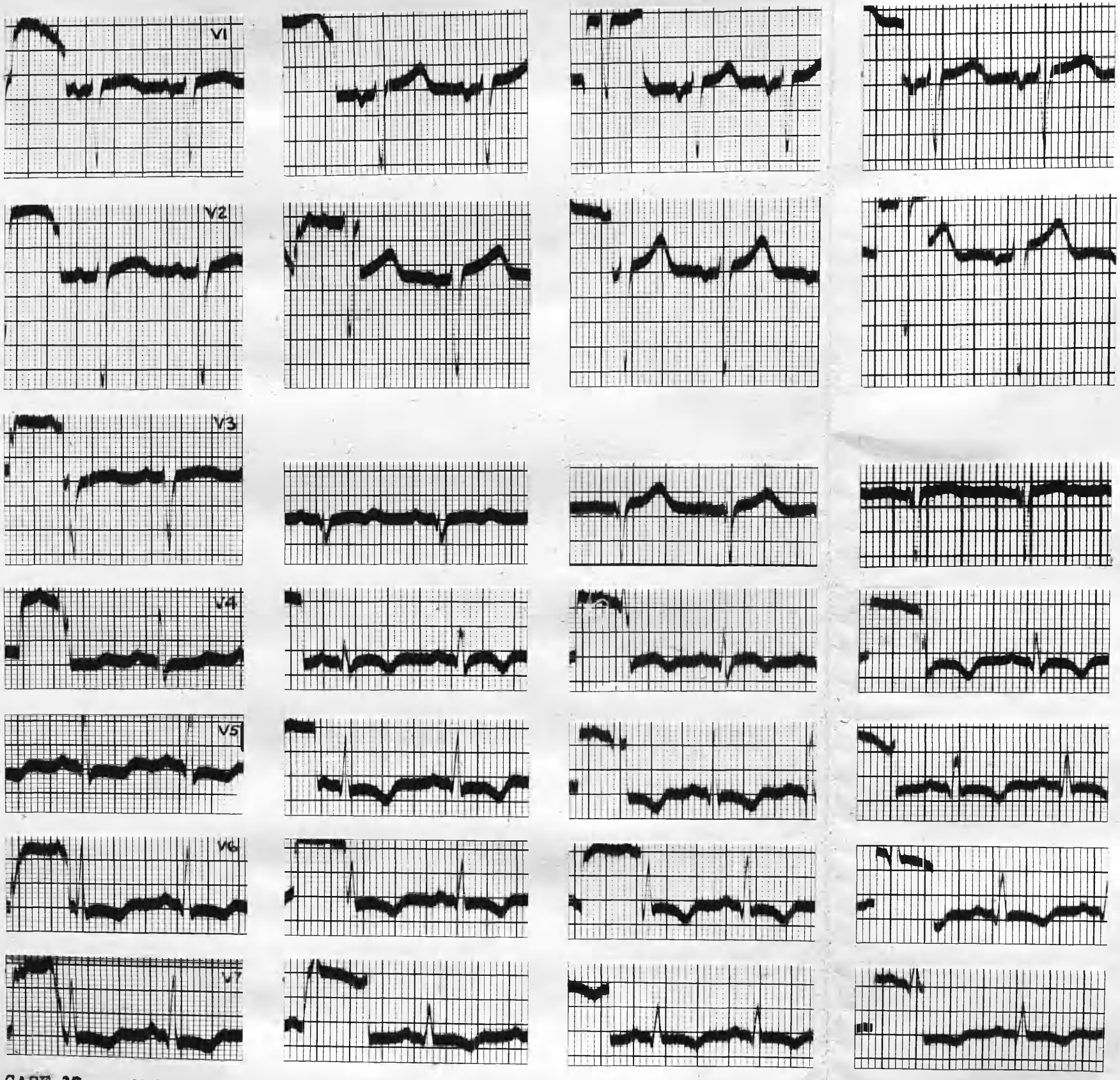


CASE 35, M.M., FEMALE. 61 YEARS.
1 WEEK
AFTER ONSET.

3½ WEEKS
AFTER ONSET.

5½ WEEKS
AFTER ONSET.

8½ WEEKS
AFTER ONSET.



CASE 35, M.M., FEMALE. 61 YEARS.

1 WEEK AFTER ONSET.

3½ WEEKS AFTER ONSET.

5½ WEEKS AFTER ONSET.

8½ WEEKS AFTER ONSET.

Case 36, J.C., male, 46 years.

This man had an attack of prolonged retrosternal pain occurring while at rest one week before admission to hospital. There was no circulatory collapse. An electrocardiogram taken nine days after onset of pain shows Q_2T_2 and Q_3T_3 patterns with elevation of the ST segment. There is confirmatory evidence of posterior infarction in lead VF. A Q wave of 3 mm. is present in lead I along with slight elevation of the ST segment. These signs in lead I raise the possibility of lateral as well as posterior infarction. Leads V6 and V5 are very similar to lead VF and indicate the lateral extension of the posterior infarction which reaches as far forward as the junction of the lateral and anterior ventricular walls. The anterior praecordial leads show changes viz. tall R, short S and prominent T waves which are due to the combined effects of two factors, viz. cardiac position and the reciprocal effects of the posterior infarction. From a consideration of the prominent R wave in lead VL, it is evident that the heart is intermediate, semihorizontal or horizontal. In the latter two positions, counterclockwise rotation of the heart is usually present and thus the anterior praecordial leads derive their potentials from the free wall of the left ventricle. The

QRS pattern which results consists of large R, short S and prominent T waves similar to that of the present case. While therefore, cardiac position may largely account for the RST configuration, the subsequent change in form of the T wave to a large symmetrical spike-like pattern suggests that it is showing the reciprocal influence of the posterior infarction. At three and a half months after onset, it has again diminished in size and is asymmetrical while the RS conflex has remained unaltered. Such fixity of the RS pattern in the presence elsewhere of very active signs of healing (to be described) is suggestive that it was derived from the free wall of the left ventricle uninfluenced by the posterior infarction whereas the slight variation in the T wave indicates that the effects of the zone of ischaemia surrounding the infarct were reciprocated in the anterior praecordial leads.

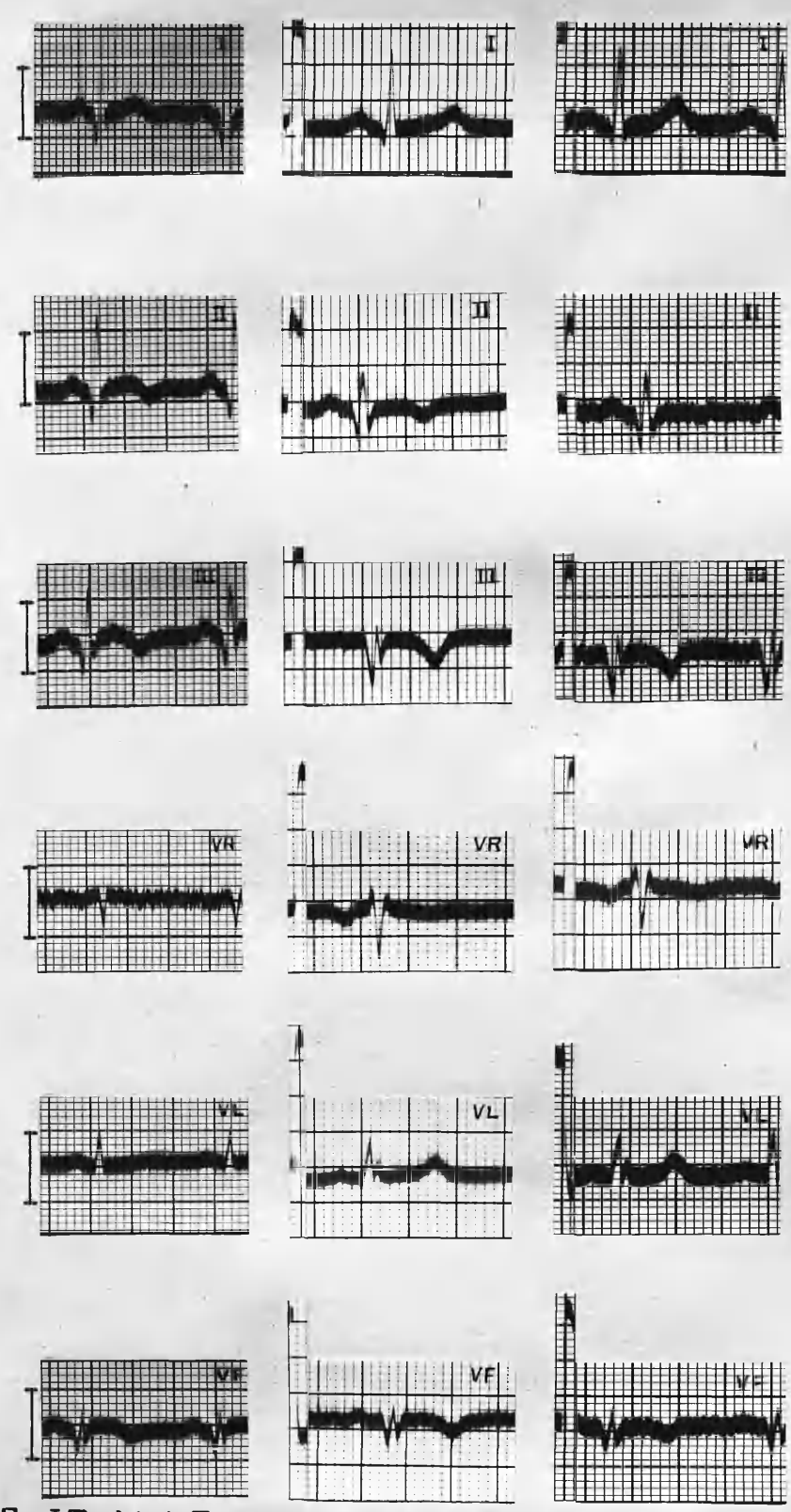
Elsewhere in the electrocardiogram at six weeks after onset, there are striking regressive changes. At this relatively early date the R wave has become very tall from lead V4 through V6 but not in lead VF. The evolution of the ST-T segment has progressed rapidly at the V4 and V5 levels. In the former the T wave now shares in the slight reciprocal exaggeration seen in the T wave

of the anterior praecordial leads; in the latter the ST-T segment is practically isoelectric. Lead V6 still displays elevation of the ST segment and inversion of the T wave in spite of the increase in the R wave. In the axillary leads there is little change in the Q waves, but the Q/R ratio has of necessity fallen owing to the increase in the R wave. It is possible, therefore, that the Q wave in these leads is partly septal in origin. These R changes clearly indicate restoration of temporarily diminished function in the apical and lateral ventricular walls. They are reflected because of the galvanometric connections invariably employed as a decrease in the R waves in leads II and III but the deflection is not affected in lead I.

The last set of electrocardiograms shows persistence of the signs of a healing posterior infarction in the standard and unipolar limb leads. The anterior praecordial leads show little change, as previously stated, but the axillary leads show further regressive signs. The ST segment is isoelectric; lead V5 now shows a prominent upright T wave and lead V6 a small upright T wave. Lead V7 taken for the first time alone shows signs consistent with an organising infarction, viz. a Q wave of 2 mm. compared with a subsequent R wave of 6 mm. and a

flat ST-T segment.

This case illustrates the rapidity with which infarcted muscle in the lateral part of a posterolateral infarction may recover. It also stresses the importance of assessing cardiac position before interpreting unusual RST forms. In this case a posterolateral infarction in a horizontal or semihorizontal heart with frank counter-clockwise rotation produced slight reciprocal effects on the T waves alone in the anterior praecordial leads, the R and S deflections of which, nevertheless, resemble those frequently produced by the reciprocal influence of a posterior infarction.

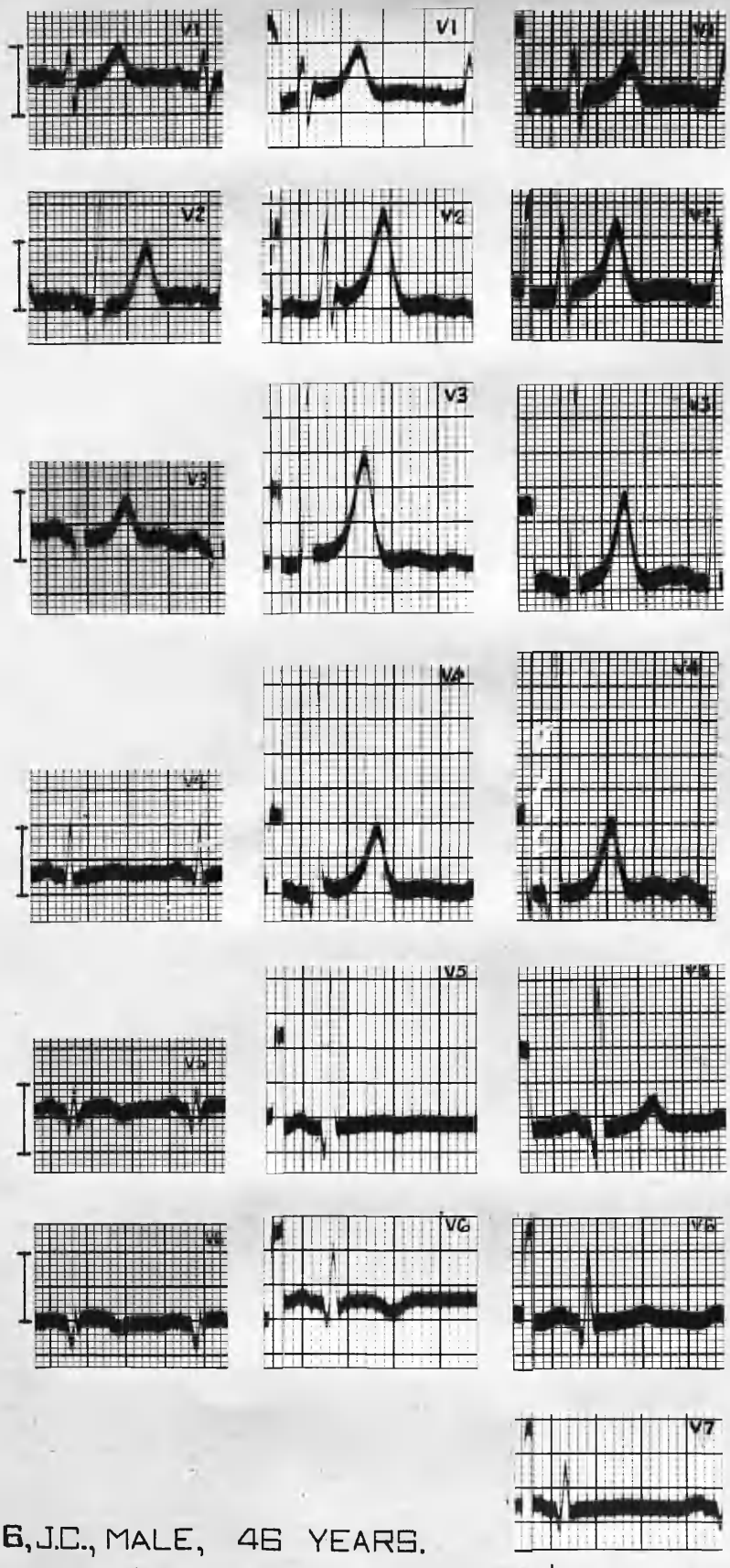


No 36, J.C., MALE, 46 YEARS.

9 DAYS AFTER ONSET.

6 WEEKS AFTER ONSET.

3½ MONTHS AFTER ONSET.



No 36, J.C., MALE, 46 YEARS.

9 DAYS AFTER ONSET.

6 WEEKS AFTER ONSET.

3½ MONTHS AFTER ONSET.

Case 37. W.A., male, 55 years.

This patient, a miner of 55 years, had his first attack of substernal pain with collapse three months before he was finally admitted to hospital. He made a fair recovery at home from the original attack and in spite of persistent dyspnoea insisted on returning to work. For the fortnight preceding admission he had recurrent burning substernal pain while at work, culminating in collapse. An electrocardiogram taken four weeks after onset of the second attack shows the typical signs of a postero-lateral infarction. In general, a posterior infarction tends to reduce forces which are antagonistic to those which produce the R wave in anterior praecordial leads; hence the prominent R waves in leads V2 and V3. Probably for the same reason the S waves in leads V1 and V2 are shortened. The axillary leads show a sharp transition from an ischaemic zone in leads V5 (and V6) to a zone with practically no functioning muscle in lead V7.

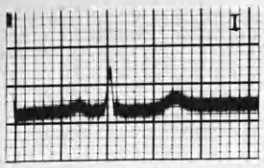
The patient again made a fair recovery and insisted on returning to work as a miner. He was seen six and a half months after onset. He admitted to only slight dyspnoea on walking a mile underground. Blood pressure, which had been at shock levels during his

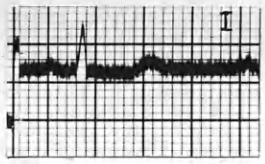
hospital stay, had risen to 130/80. The cardiac sounds were of indifferent quality. An electrocardiogram shows persistent signs of posterior infarction with the QRS deflection of leads II and III now broadened and splintered. The praecordial leads show diminution in the height of the R wave in leads V2 and V3 - the posterior infarction no longer reducing forces antagonistic to the R wave in these leads. At the same time the S wave in leads V1, V2 and V3 has increased in depth and the ST segment previously iso-electric has resumed its normal slightly elevated level. The changes reciprocal to posterior infarction in these leads have entirely disappeared indicating healing in a particular part of the infarct. The increase in depth of the S wave in the anterior praecordial leads has not been accompanied by a heightening of the R wave in the apical and anterior axillary leads because of the close proximity of the lateral edge of the infarct. At the same time the ST segment of leads V4 and V5 still shows slight depression which is the well known reciprocal effect of a recent posterior infarction and which was originally shared by all the anterior and the apical leads. The axillary leads also show that there is still severe involvement of the left ventricular wall in the V7 position although there is a slight increase in

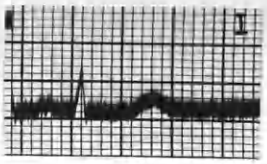
the positivity of the small R wave and although the zone of ischaemia has receded. Thus the signs of restitution in the anterior praecordial leads noted above cannot be ascribed to any healing in the lateral part of the infarct. The form of these leads at no time suggested infarction of the interventricular septum, so that the healed area has been in the more medial parts of the posterior ventricular wall. Some indication of the large size of the area left unhealed, however, may be gained from a study of lead VF. During the acute stage a small splintered QS deflection was present indicating a transmural infarction. Subtending, as it does, a large area of diaphragmatic ventricular surface lead VF soon recovers a positive R wave in most cases but six and a half months after onset only a small slurred R wave has emerged. Confirmation was obtained radiologically; "There was fairly marked enlargement of the left ventricle. On screening, there was relative loss of expansile pulsation in the diaphragmatic aspect of the left ventricle".

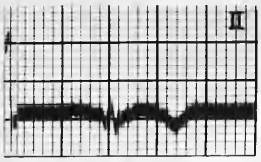
A follow-up enquiry revealed that this patient died of congestive cardiac failure about a year later.

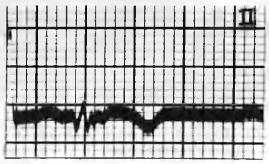
From the electrocardiographic point of view, the case is an example of partial healing; one edge or segment of the infarct heals satisfactorily but elsewhere there is widespread patchy fibrosis.

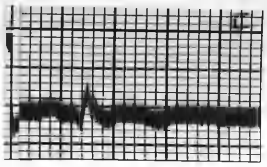


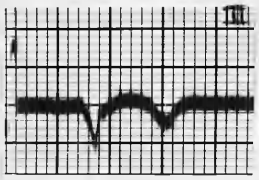


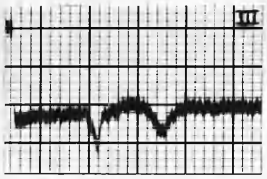


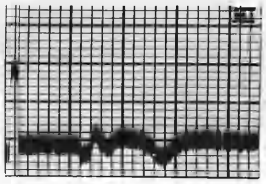


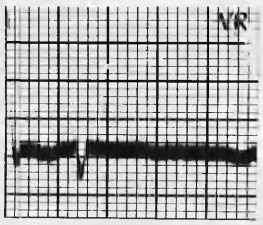


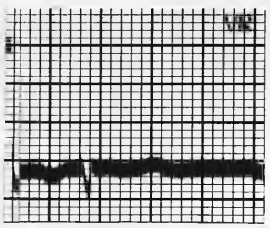


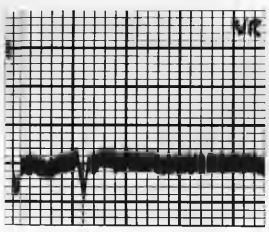


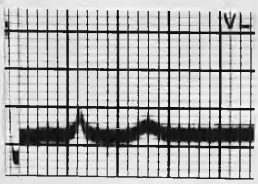


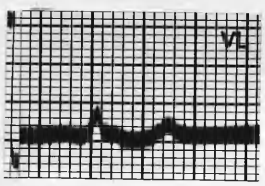


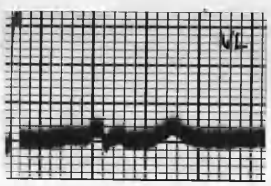


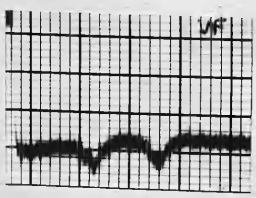


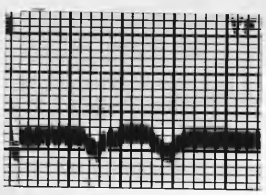


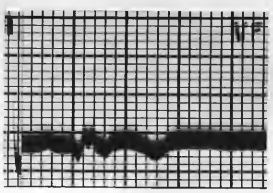










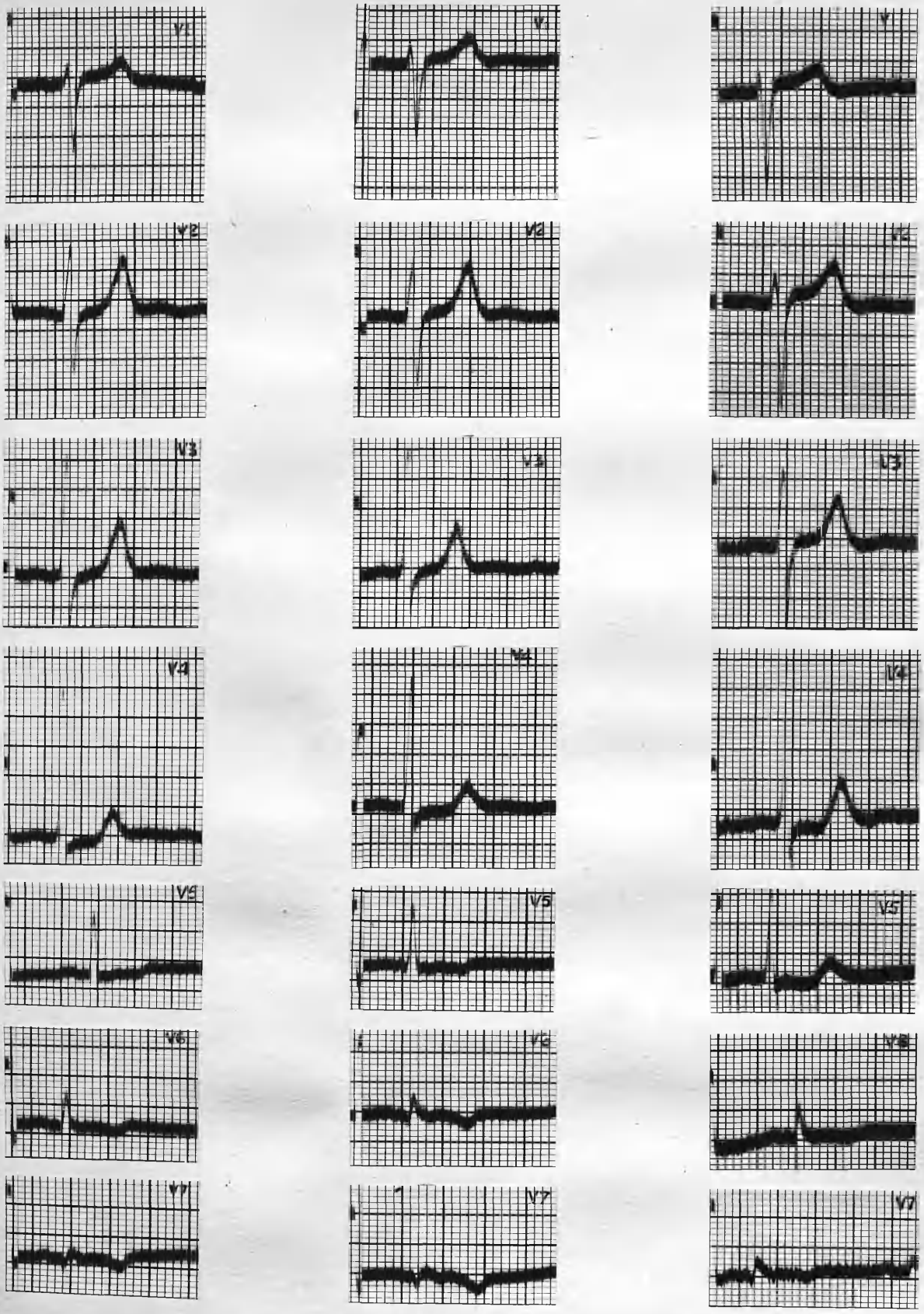


CASE 37, W.A. MALE, 55 YEARS.

4 WEEKS AFTER ONSET.

6 WEEKS AFTER ONSET.

6½ MONTHS AFTER ONSET.



CASE 37, W.A. MALE, 55 YEARS.

4 WEEKS AFTER ONSET.

6 WEEKS AFTER ONSET.

6½ MONTHS AFTER ONSET.

Case 38. A.M., 59 years, male.

This patient, an engineer of 59 years, had had exertional angina for several months culminating in a prolonged attack of crushing pain, which was accompanied by some collapse. The first electrocardiogram was not obtained until two months after onset, when he attended the out-patient department. It shows the typical signs of an organising posterior infarction in leads II and III. Lead VF is confirmatory. There is a very small Q wave in lead I, not in itself sufficient to raise the possibility of lateral infarction. The anterior praecordial leads show the changes reciprocal to posterior infarction, viz; some exaggeration of the R and T waves. There is also severe shortening of the S waves to such a degree that it may be presumed that they were small even previous to infarction because of counterclockwise rotation of the heart around its long axis; thus the epicardial surface of the left, not the right, ventricle is presented to the electrode in the V2 and V3 positions as well as in the apical and axillary leads. Lead V6 shows a sudden fall in the amplitude of the R wave compared to that of lead V5. The Q wave is relatively prominent in lead V6, measuring fully 2 mm. which is 50% of the succeeding R wave and occupying .02 sec; the ST-T segment has become suddenly

flat. It is clear that lead V6 overlies the organising edge of the infarct which is at this point subendocardial. Lead VL shows no sign of this lateral extension of the infarct in spite of counterclockwise rotation which favours the reference of potential variations of the lateral wall to the left arm. Presumably the infarct involved the posterolateral rather than the anterolateral wall from which lead VL is clearly deriving its potentials.

The patient was examined a year later. He had returned to work as an engineer. He complained, however, of a gripping feeling in the chest on exertion without shortness of breath. There was very slight facial cyanosis. B.P. was 120/90. The chest was of long type and the cardiac sounds were of medium intensity and pure. An electrocardiogram shows the persistent signs of an organising posterior infarction in the standard limb leads. Confirmation is seen in lead VF. In addition lead VL now shows a Q wave of 1.5 mm. or approximately 25% of the succeeding R wave; from onset to nadir it occupies fully .02 sec. and it is followed by a slurred R wave. It is clear that lead VL now reflects potentials from the subendocardial infarction of the lateral wall. Either a slight shift in cardiac position has occurred to bring the

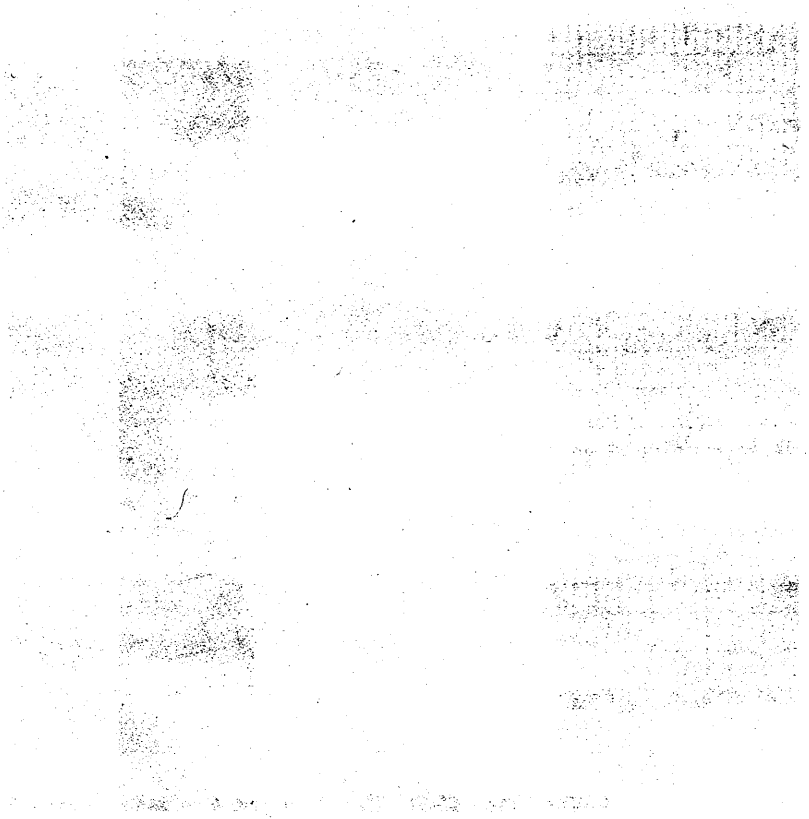
infarction into the region subtended by the left arm or in the process of healing there has been some extension of fibrosis to involve the anterolateral as well as the posterolateral wall although the latter is unlikely in view of the relative fixity of the axillary leads.

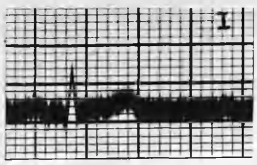
Lead VR exhibits as before an rS form derived from the epicardial surface of the right ventricle. Thus the borderline Q wave in lead I is attributable to the initial positivity of the right arm, which is physiological, just as much as to the initial negativity of the left arm and left leg which are due to the infarction.

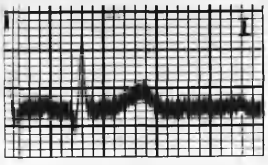
Of the praecordial leads, lead V4 shows a great increase in the height of its R wave. Corresponding increments are seen in lead V3 forwards, but not in the three axillary leads owing to their proximity to the infarct. These large R waves are accounted for largely by a full return of function in the healthy areas of the left ventricular wall. They are associated with normal ST segments and T waves and with the return of S waves of moderate length in the anterior praecordial leads. This is in contrast with the long deep S waves commonly found in these leads in healed posterior infarcts. The difference is due to the counterclockwise rotation of the heart present in this case such that the epicardial

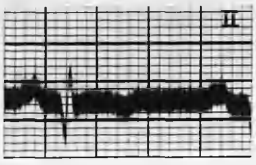
surface of the left ventricle and not the right is the chief determinant of the pattern in leads V1, V2 and V3. This relationship also explains the increase in the R waves in leads V1 and V2 which in healed posterior infarcts ordinarily show a decrease in height. Lastly the question arises as to whether additional factors are concerned in the production of these large R waves. There may be a minor degree of left ventricular hypertrophy insufficient to affect the ST and T segments but more probably there is a passive enhancement of the potentials of the anterior left ventricular wall due to scar tissue in the region of the infarct - a sequel of the early reciprocal exaggeration of the R waves. However, the T waves are not affected. The presence of the scar tissue in the posterolateral wall is documented by the large Q of lead VF. the large Q and delayed R waves of leads V6 and V7 and the significant Q and slurred R waves of lead VL. This case is an example of a fixed healed posterolateral infarct in a horizontal heart with moderate counterclockwise rotation. The ultimate electrocardiographic pattern is influenced by the residual fibrosis on the one hand and the compensatory functional recovery on the other. That such a development is effective to a certain extent is seen by

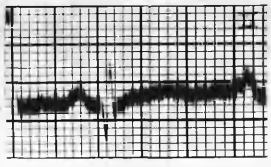
the fair measure of health which the patient enjoyed. Radiologically there was no cardiac enlargement and no area of diminished pulsation.

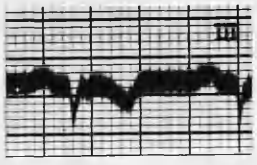


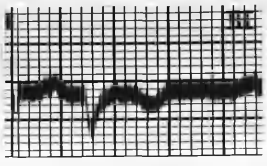


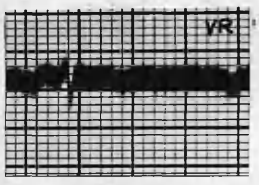


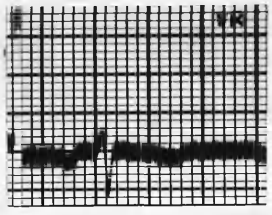


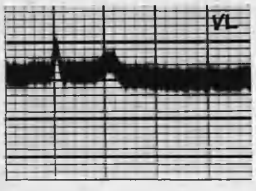


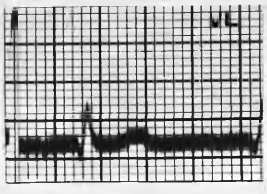


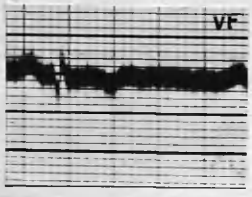


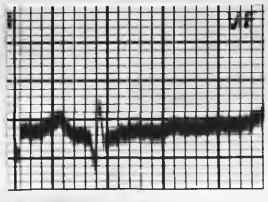








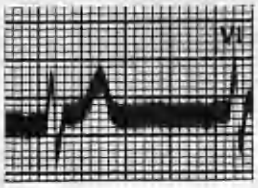


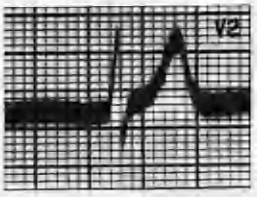


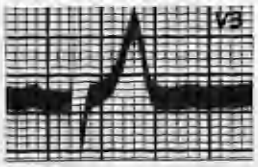
CASE 38, A.M. MALE, 59 YEARS.

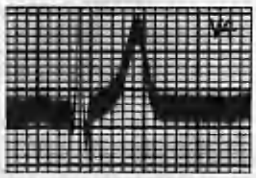
2 MONTHS AFTER ONSET.

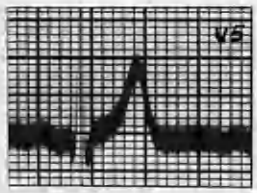
15 MONTHS AFTER ONSET.

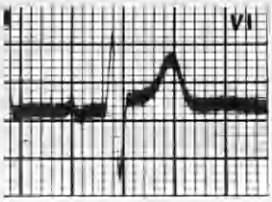


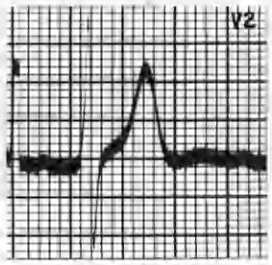


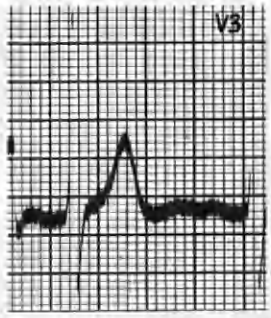


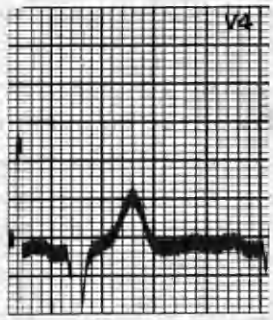


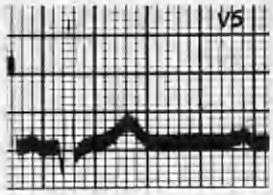


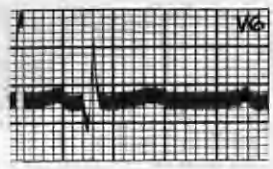


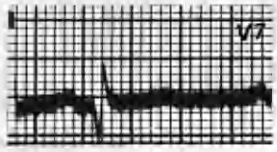








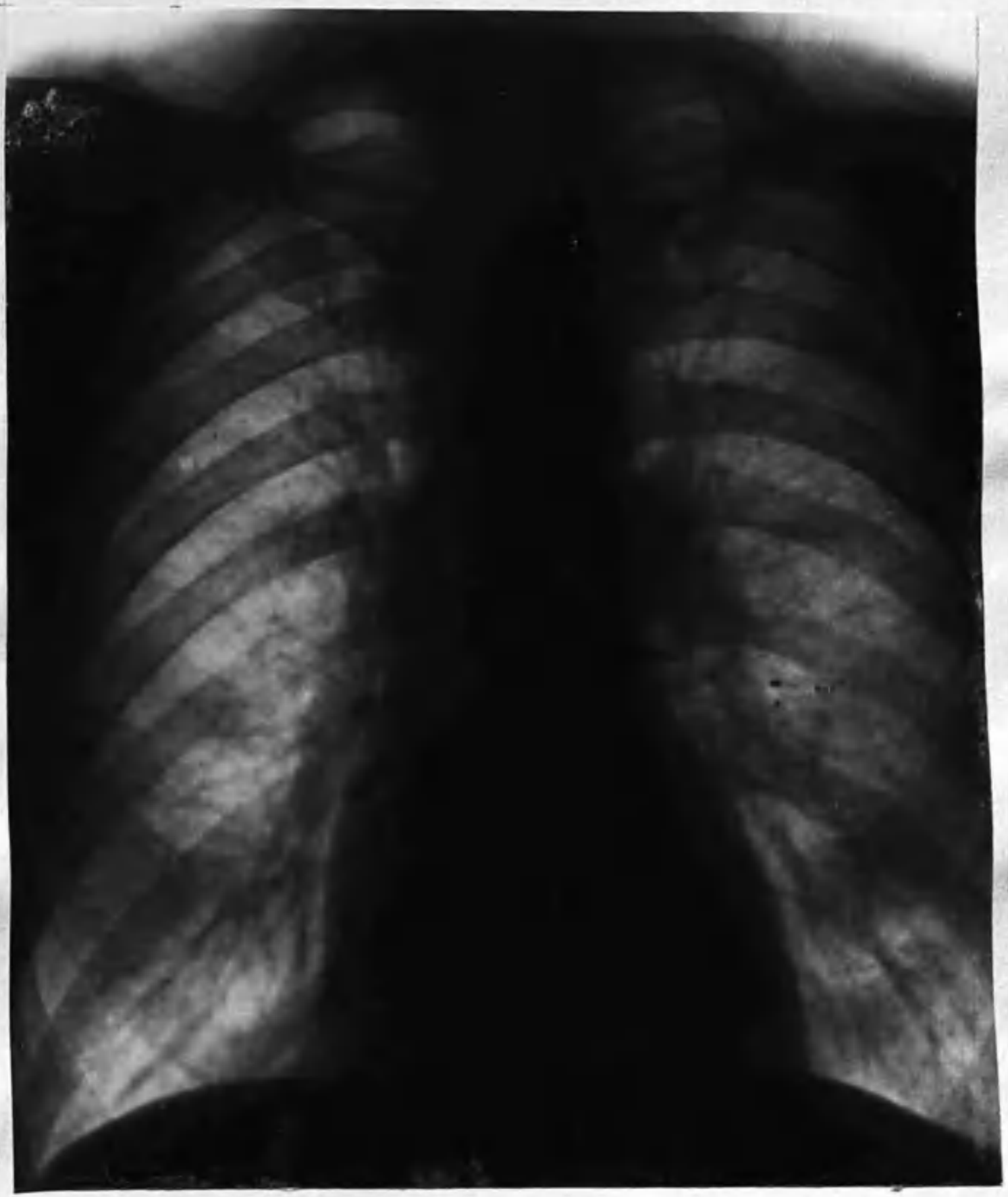




CASE 38, A.M. MALE, 59 YEARS.

2 MONTHS AFTER ONSET.

15 MONTHS AFTER ONSET.



ANTERO - POSTERIOR

Case 39 A. A. M., male, 54 years.

This patient, a commercial traveller of 54 years, had a prolonged attack of substernal pain while at work one week before the first electricardiogram was obtained. The prominent R of lead VL indicates that the heart is either horizontal, semi-horizontal or intermediate in position. The pattern of the ST-T segment of lead VF is typical of early infarction. Its QRS complex is also attributable to infarction. If on the one hand the heart is horizontal, then the infarct is probably situated in the septum, the Q wave representing initial negativity of the right side of the septum, either because of a complete transeptal lesion that serves as a window capable of transmitting negative potentials from the left to the right ventricular cavity or because of a lesion confined to the left side of the septum that results in the activation of intact septal remnants from the right rather than the left Purkinje network. Furthermore, the presence of a RS complex in lead VF may signify that the epicardial surface of the free wall of the right ventricle is influencing the potentials of the left leg, a further indication that the heart is horizontal. On the other hand, if the

heart is intermediate the QRS complex in lead VF is attributable to an incomplete transmural infarction of the posterior wall of the left ventricle. The latter supposition is favoured by examination of lead V6 which, by virtue of its domed ST and shallow inverted T suggests that it overlies the epicardial edge of the infarct. The Q wave of this lead is equivocal and its subsequent fate is against it being significant in retrospect. In so far as there are signs of epicardial involvement in lead V6, the infarct was probably posterior rather than septal. The distinction is to a certain extent academic for most large infarcts in this region involve both the posterior and the septal walls of the left ventricle and the subsequent development of this infarct favours this view.

The anterior praecordial leads show unduly prominent T waves; the R waves, however, are not exaggerated, but there is shortening of the S waves which is commonly found in the anterior praecordial leads in the presence of posterior infarction. During his first two weeks in hospital, the patient remained sharply ill. He had several attacks of praecordial pain requiring morphine for their relief. The next electrocardiogram

taken twelve days after onset shows significant changes. There is an increase in positivity of the T waves of the praecordial leads including leads V5, V6 and also in lead VL, the extent of which is too great to be explained entirely as the reciprocal of the original infarction. Combined with the new ST depression in leads V5, V6 and VL and the increase of the R waves of leads V1 and V2, these changes indicate further acute injury to the posterior wall. In addition an abnormal slurring or notching has developed in the terminal portion of the QRS complex of leads V1 through V3 suggesting a lesion in the last portion of the heart to become activated viz. the posterobasal wall of the left ventricle. The Q waves of leads V4 to V6 have disappeared indicating diminution of conduction in the left side of the septum and the intrinsic deflection of lead V1 is delayed. These signs indicate that the further acute injury has been posteroseptal and also posterobasal in situation causing incomplete left bundle branch block with some focal conduction defect in the posterobasal wall of left ventricle. Lastly, the pattern of the P waves has been severely disturbed in this electrocardiogram; the praecordial leads and lead

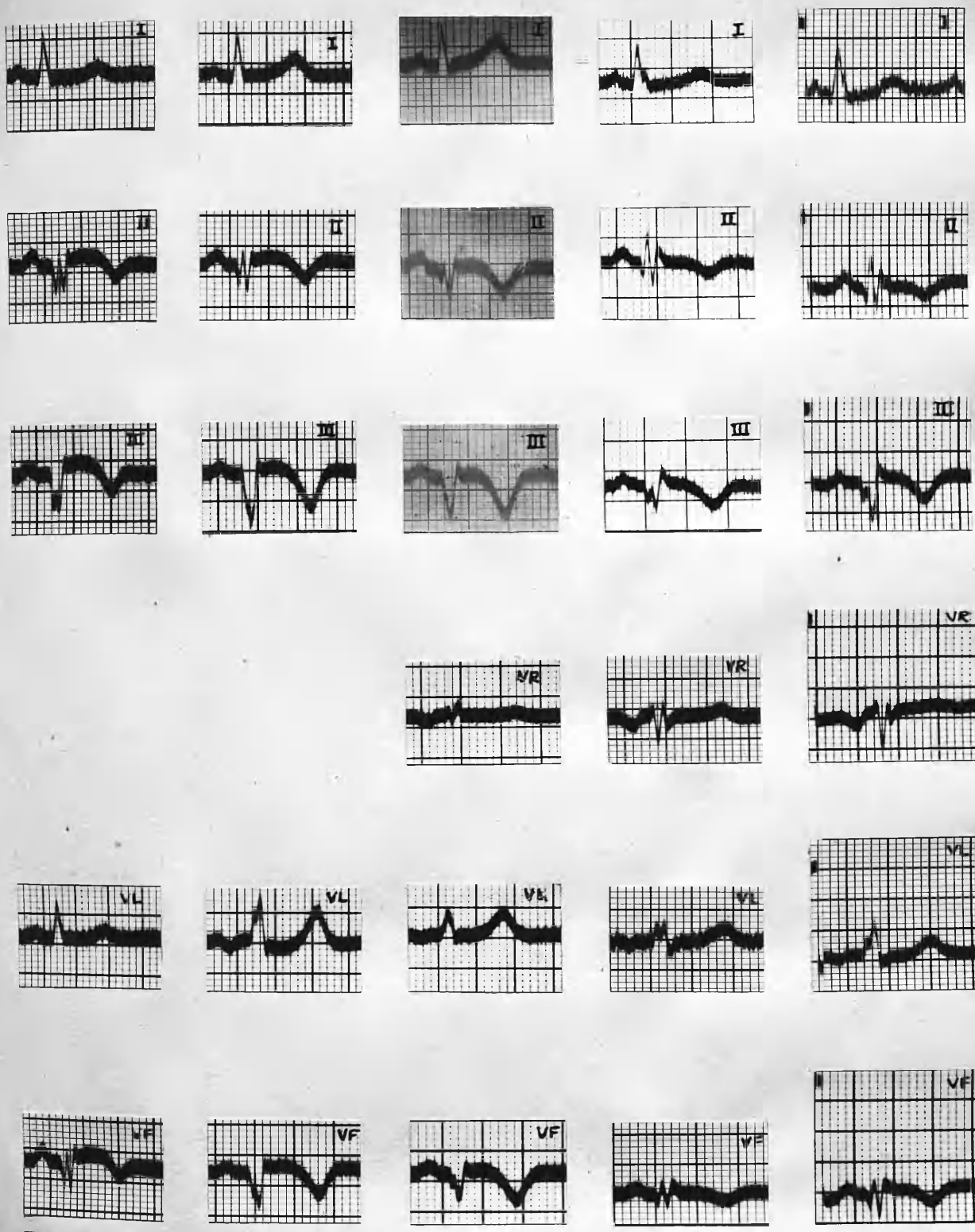
VL show inverted or diphasic P waves but the PR interval is not affected. Hence it is probable that this large posteroseptal infarct also involved the auricular wall.

An electrocardiogram taken a week later shows some resolution of the foregoing lesions. The ST depression disappears from leads V5, V6 and VL and their T waves revert to their former size. The slurring of the QRS complex in the anterior praecordial leads is much less prominent although they are still unduly broad. The Q wave of lead V6 has returned and the intrinsic deflection in lead VL is less delayed. The incomplete left bundle branch block would appear to have been temporary and any focal conduction defect has become less definite.

The patient was allowed home at the end of three months. He was not seen again until the fifteenth week after onset. Standard limb leads now show the fixed pattern of posterior infarction. The duration of QRS has lengthened and now measures .10 to .12 sec. in standard and unipolar leads. A return of left bundle branch block is excluded by the combination of initial negativity of the left ventricular cavity as indicated by the Q waves in leads V5 and V6 with initial

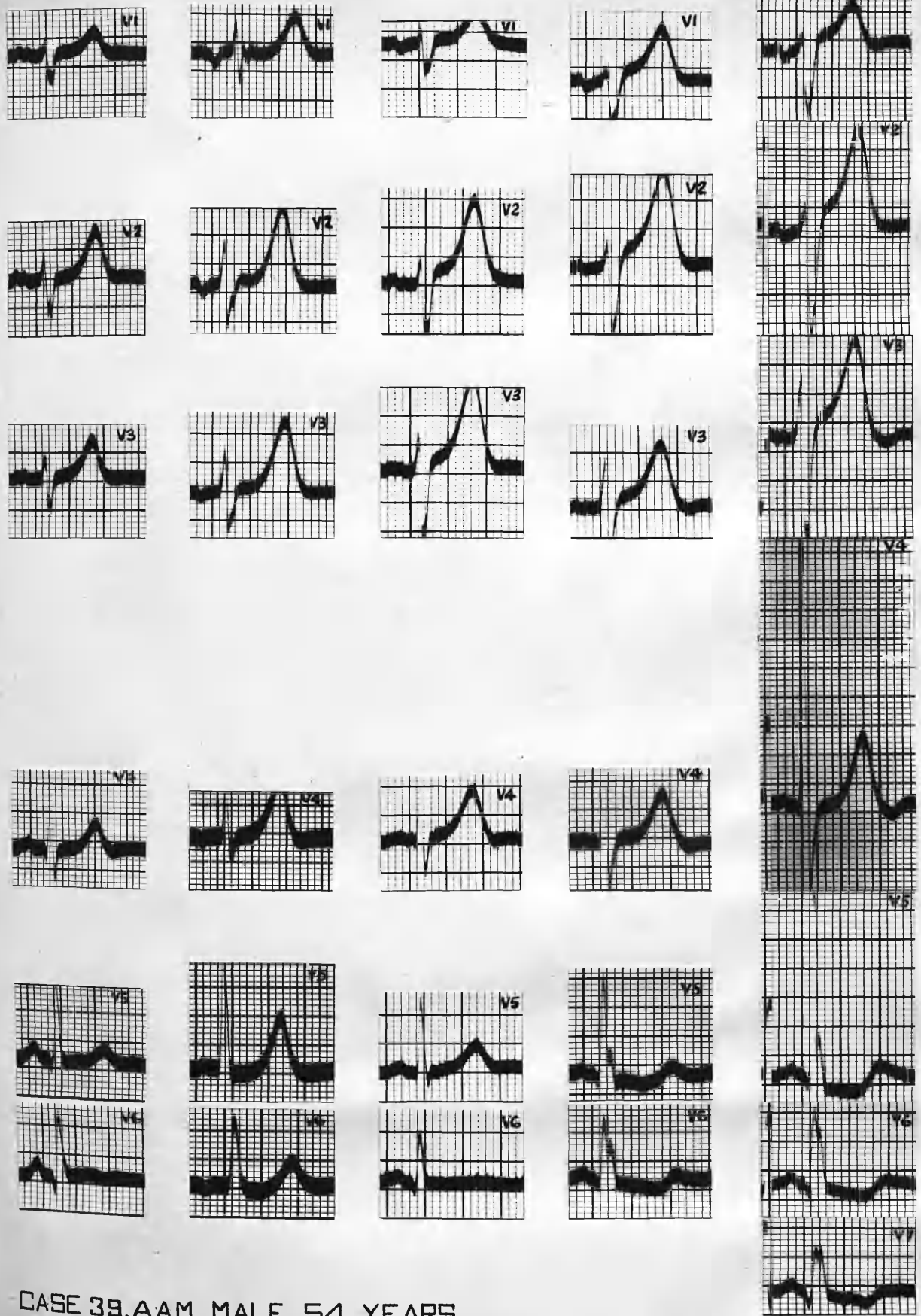
positivity of the right ventricular cavity indicated by the small R in lead VR. Reciprocal changes persist in leads over the right side of the praecordium in so far as the T waves remain very tall. Any exaggeration of the R waves has disappeared and coincidently the S waves have lengthened. The R waves in leads V4 and V5 show a great increase in height. Such an abrupt increase between two adjacent leads in the size of a deflection, invariably the R or S wave, which is of an order well outwith the physiological, is a common feature in healed infarctions and suggests a sudden local change in the functional capacity of the subjacent muscle. In this case, the very tall R in lead 4 may be due to ablation of all forces antagonistic to the R wave such as would arise if some part of the posterior left ventricular wall were totally fibrotic, or, alternatively, compensatory hypertrophy of the relatively sound anterior parts of the left ventricular wall may contribute to the height of this R wave. Leads V5 and V6 show further local change; QRS is .12 sec. broad and the R wave shows a prominent notch on its descending limb, indicating a defect in conduction in the free lateral wall of the left ventricle. Lead V7

taken for the first time shows a Q/R ratio of 25% due to its proximity to the infarcted region. The sagging ST and diphasic T pattern in these leads is secondary to the conduction defect. This case shows clearly the value of the unipolar praecordial leads in detecting spread of infarction and in assessing the character and localisation of the residual injury following infarction which does not declare itself by any fresh change in the limb leads, standard or unipolar.



CASE 39, A.A.M. MALE, 54 YEARS.

7 DAYS AFTER ONSET.	12 DAYS AFTER ONSET.	19 DAYS AFTER ONSET.	15 WEEKS AFTER ONSET.	20 MONTHS AFTER ONSET.



CASE 39, A.A.M. MALE, 54 YEARS.

7 DAYS AFTER ONSET.	12 DAYS AFTER ONSET.	19 DAYS AFTER ONSET.	15 WEEKS AFTER ONSET.	20 MONTHS AFTER ONSET.

Case 40. J.M., 50 years, male.

This patient had a severe attack of praecordial pain accompanied by shock on the morning of admission to hospital. Blood pressure was 108/72 falling to 90/60 on the third hospital day. During the first fortnight, there was basal pulmonary congestion and three weeks after onset another attack of chest pain occurred situated substernally and over the right front. The differential diagnosis lay between further infarction and pulmonary embolism although neither haemoptysis nor pleural friction were present to substantiate the latter. The patient eventually left hospital after an eight weeks' stay. He was symptom-free, but the cardiac sounds were still poor and the blood pressure 90/70.

The first unipolar electrocardiogram was not obtained until sixteen days after onset. The relatively small R and deep S waves in leads V1 through V5 indicate that they reflect the potential variations of the right ventricle; the transitional zone is situated at position V5 and lead VL shows an RS complex. These facts indicate a vertical to semi-vertical position of the heart. Lead VF (not obtained until later) reflects therefore the potential variations of the free posterior

wall of the left ventricle. It shows a small QR deflection less than five millivolts in amplitude. In spite of its small size, its association with a shouldered ST segment and inverted T wave, together with the classical signs in leads II and III, leave little doubt that there is an infarction of the posterior wall of the left ventricle. The praecordial leads sixteen days after onset show no significant abnormality, but four weeks after onset or one week after the second attack of chest pain, there are fresh changes in the praecordial leads over the right side of the praecordium and in lead VR. In lead V1 the small R deflection has disappeared, ST is domed and T inverted; in lead V2, R is smaller than in the previous tracing and T is of shallow inverted type and in lead V3, T is practically flat. Lead VR shows no change in its QR complex, but ST is now a plateau and T inverted. In explanation of these changes, several possibilities were considered: (1) Shift of cardiac position so that the potential variations of the posterior wall of left ventricle are transmitted more readily to the right praecordium but no concomitant change in QRS has occurred to support this theory: (2) Pericarditis but this is unlikely in so far as the

main changes are confined to two or at most three leads, and the apical and left praecordial leads are not affected; (3) Fresh infarction of the free anterior wall of the right ventricle; this implies a second infarct separate from the first and infarction of the right ventricle is acknowledged to be a rare condition. Furthermore, an infarction in this position should not have obliterated the early R wave of lead V1 due, as it is, to initial positivity of the right side of the inter-ventricular septum; (4) Spread of the original infarction forwards into the septum so that the newly infarcted area would act as a window capable of transmitting the negative potentials of the left ventricular cavity to the right and thus to the V1 position; (5) Acute right ventricular dilatation occurring one week after pulmonary infarction. The absence of clinical signs to suggest isolated right heart failure is a priori against this explanation although it can account for the new ST and T signs especially since they are greatest in lead V1. It does not explain the absence of the R wave in lead V1. The fourth possibility, viz; spread of infarction into or farther into the septum appears to be the likeliest interpretation.

Electrocardiograms at six and a half and at nine weeks reveal a speedy return to the previous pattern in leads V2 and V3, but lead V1 remains abnormal with its absent R, domed ST and inverted T and in lead VR, T becomes almost flat again. It is noteworthy that the standard limb leads, lead VL and the leads over the left side of the praecordium remain unaffected by the spread of infarction into the septum.

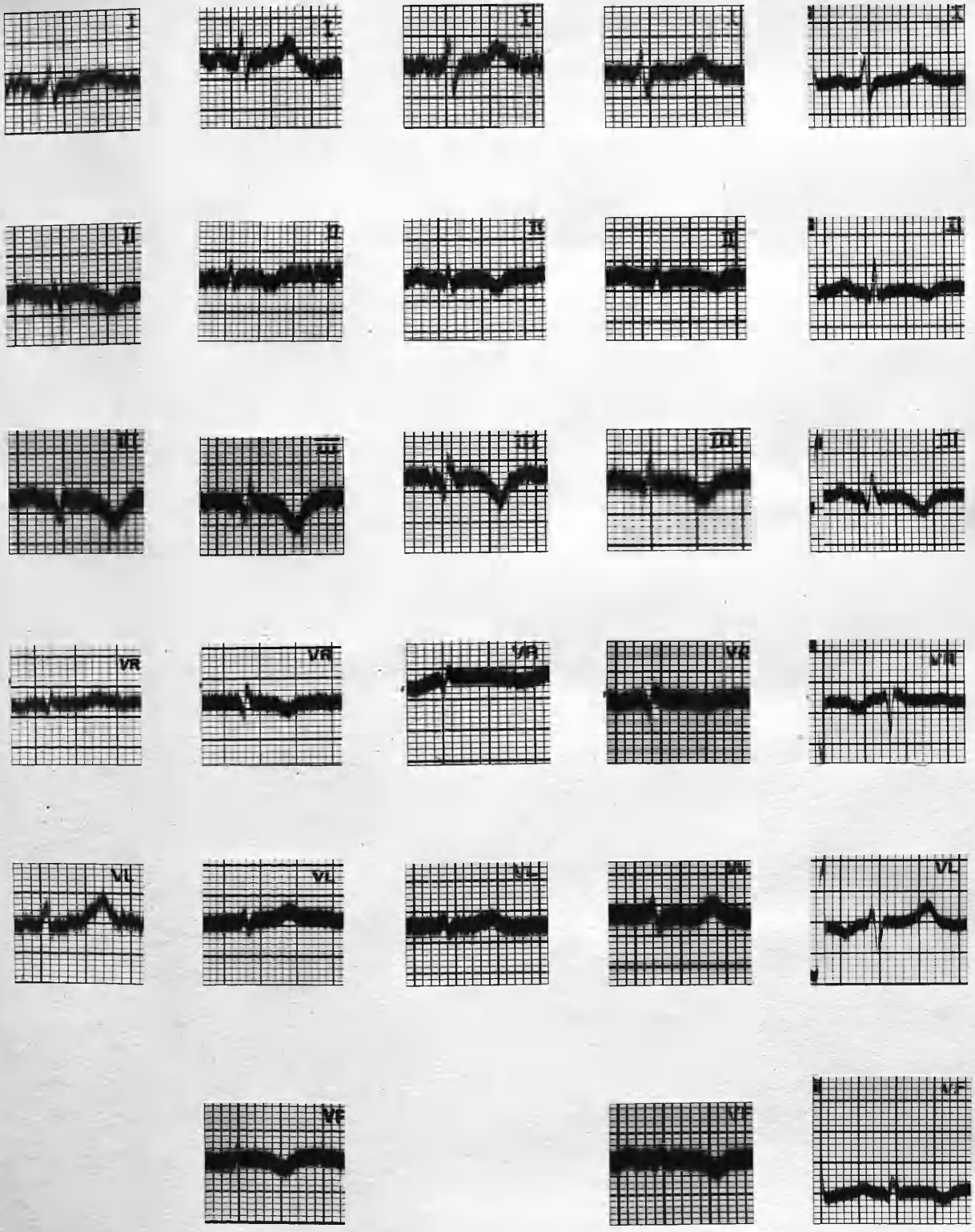
Patient was not seen again until the expiry of a year from onset. He complained of dyspnoea on climbing stairs, but not on level ground and he had occasional slight pain in the left chest. There was no clinical enlargement of the heart. The cardiac sounds remain soft and blood pressure was at its previous low level, viz; 90/70. Radioligical examination showed slight prominence of both left and right ventricles.

An electrocardiogram at this date shows the fixed pattern of posterior infarction in lead VF carried over into leads II and III. Leads VR and VL show increase in their S waves and in lead VR the ST segment is no longer domed nor is T inverted. These leads now present the signs commonly found in normal vertical or semi-vertical hearts. Reflecting, as they do, the potential variations

of the ventricular cavities, the increase of the S wave is either a favourable sign of some restitution of ventricular activity or an indication that the electrical axis is even more vertical than before. Of the praecordial leads, lead V1 like lead VR shows a return to normal of the ST segment and T wave and in addition it shows a return of the R wave. Leads V2 and V3 are again normal. This reversion to a normal pattern in leads V1 through V3 indicates that the extension of the infarct into the septum has healed with restoration of function. The axillary leads V6 and V7 are abnormal. The transitional zone has shifted further to the left and is now at V6 and the R wave in V7 is unduly small. However, there is no previous lead V7 for comparison. Although it is tempting to postulate poor muscle in the lateral wall of the left ventricle yet physiological considerations e.g., the electrical axis of the heart being vertical, the unknown thickness of tissue between the left ventricular wall and the electrode in the V7 position, account largely for variations in the height of R in this lead. Hence its significance here must remain open to doubt.

This case is an example of complete regression of certain signs in the right praecordial leads and in lead

VR and if the interpretation of the original signs offered is correct, viz; extension of infarction forwards in the septum, then the presumption is that healing and restoration of function occurred in this area. This case presents no signs of restitution of, or compensation for, the posterior part of the infarction, due probably to the fact that the praecordial leads were apparently not in a favourable position, from the outset, for recording reciprocal changes caused by the infarct.



CASE. 40,
J.M. MALE, 50 YEARS.

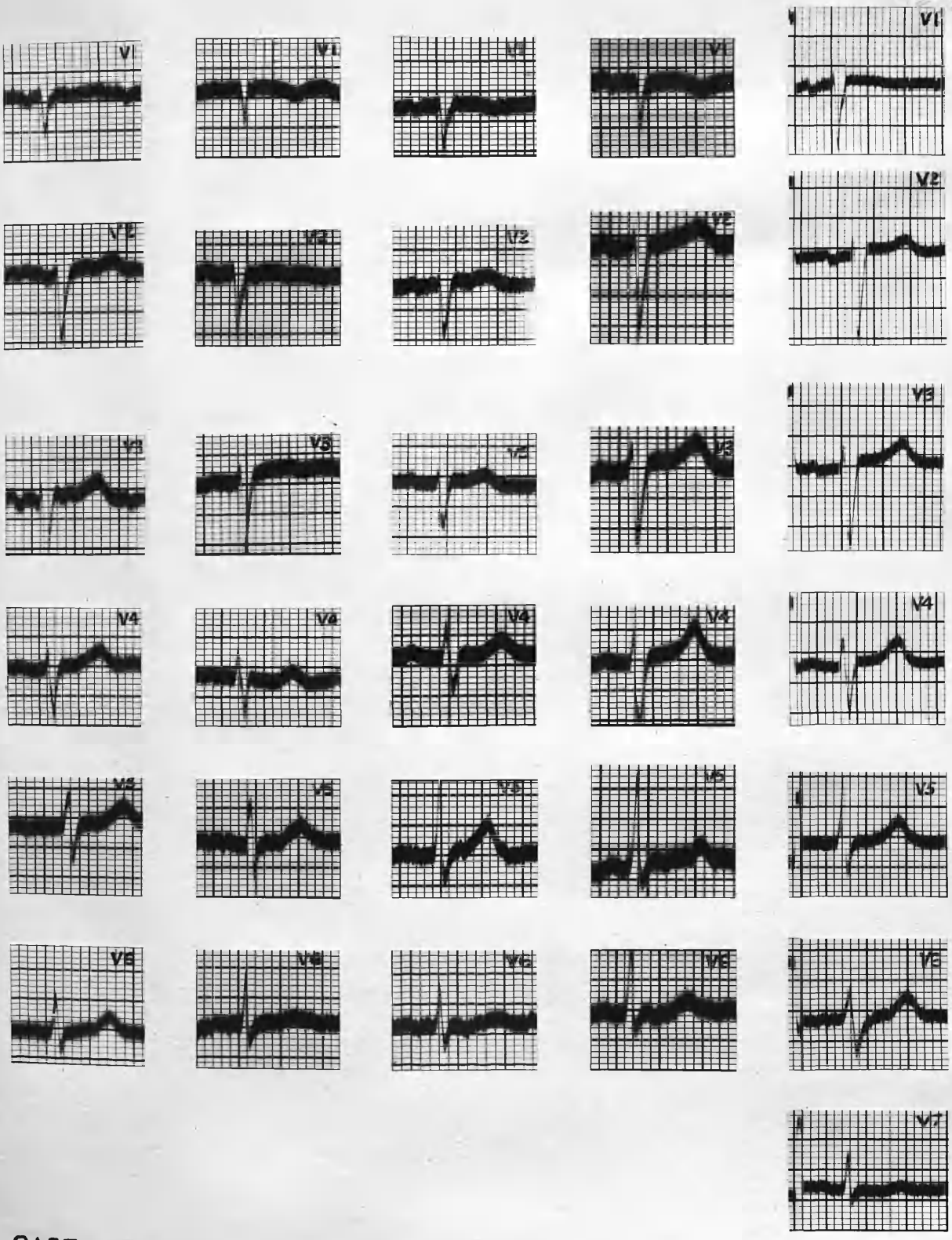
16 DAYS AFTER ONSET.

4 WEEKS AFTER ONSET.

6½ WEEKS AFTER ONSET.

9 WEEKS AFTER ONSET.

1 YEAR AFTER ONSET.



CASE. 40,
J.M. MALE, 50 YEARS.

16 DAYS AFTER ONSET.

4 WEEKS AFTER ONSET.

6½ WEEKS AFTER ONSET.

9 WEEKS AFTER ONSET.

1 YEAR AFTER ONSET.

Case 41, H.M., male, 51 years.

Patient was admitted to hospital on account of a severe attack of anginoid pain lasting several hours and accompanied by a moderate degree of shock. It was known that he had had hypertension and exertional angina for several years. Blood pressure fell from 200/125 to 164/84 during the first twenty-four hours of his hospital stay.

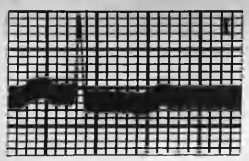
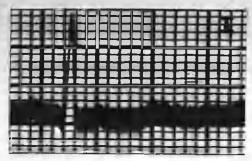
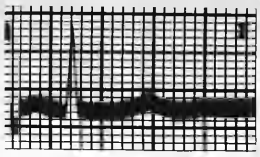
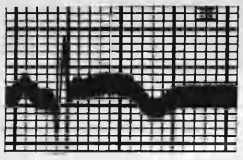
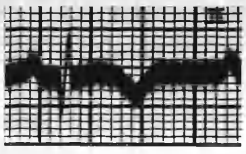
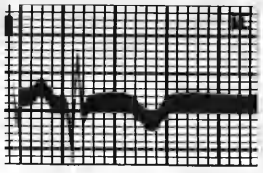
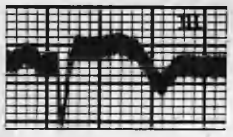
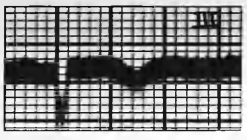
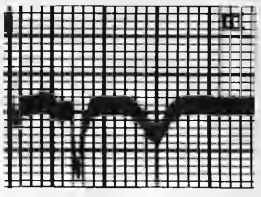
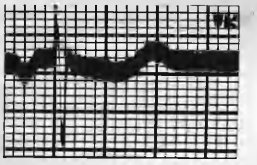
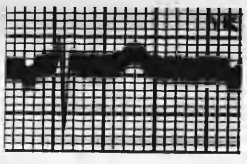
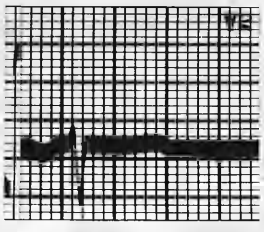
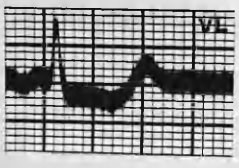
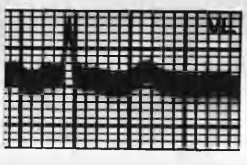
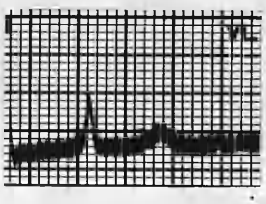
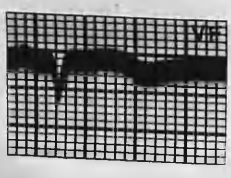
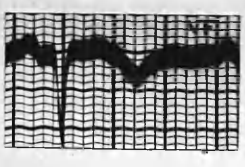
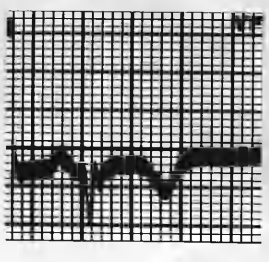
The first electrocardiogram taken twenty-four hours after onset displays elevation of ST in leads II and III and in lead VF and depression of ST in lead VL. Already T is sharply inverted in leads II and III but it is possible that T wave changes existed previously in view of the long history of exertional angina. The high R wave in lead VL indicates that the electrical position of the heart is horizontal, semi-horizontal or intermediate. In horizontal or semi-horizontal hearts, a QS complex may be recorded in lead VF as a normal variant but its association with elevation of ST in the case under discussion demands another explanation. In horizontal or semi-horizontal hearts, a QS complex in lead VF may be due to infarction of the posterior part of the interventricular septum and in intermediate hearts

(as also in semi-vertical and vertical hearts) the same recording may result from a large transmural infarction of the free posterior wall. In both, the associated ST pattern is that of infarction. Myers has pointed out that in the latter case the QS complex if often a transient finding observed during the acute stage and is replaced by a QR complex as the lesion heals. In the present instance a single QS deflection is still present five weeks after onset, hence favouring derivation of the persistent QS deflection from an infarction of the posterior part of the septum. On the other hand there is a 2 mm. depression of the ST segment in praecordial leads V2 through V6 which is reciprocal to acute posterior infarction not necessarily confined to the septum. However, the question is somewhat academic since large transmural infarcts of the free posterior wall almost always extend into the septum and infarcts of the posterior part of the septum continue into the free posterior wall so that the probability is that this patient had a postero-septal infarction.

By five weeks after onset, elevation of the ST segment has disappeared from leads II and III and lead VF and its reciprocal depression from other leads.

At seven and a half months after onset healing is well advanced. The standard limb leads and lead VF show the pattern of a healed fixed posterior infarct. The praecordial leads show a striking increase in height of R in lead V3 through V6, in leads V5 and V6 the ST segment sags slightly and T is low upright or of shallow diphasic type. The wave Q reappears in lead V4 as a very small deflection but it is 2 mm. in lead V5 through V7. Its association with a high undelayed R wave indicates that it is a reconstituted septal Q wave. The R, ST and T signs indicate ventricular hypertrophy which was probably present before infarction took place. The resurgence of these signs is attributable to the satisfactory healing of the infarct which appears to have left no residue in the praecordial leads.

Clinically the patient made a good recovery and seven and a half months after onset he had already resumed work as a clerk. Dyspnoea only occurred on walking quickly and tightness in the chest was only felt on climbing hills. Blood pressure had risen to 172/100. The early left ventricular hypertrophy is confirmed in the radiological miniature of the same date.

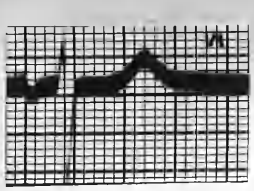
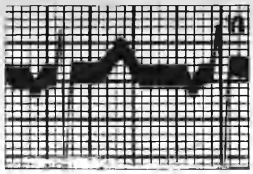
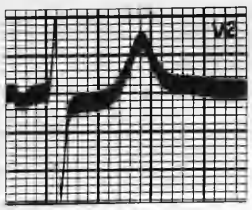
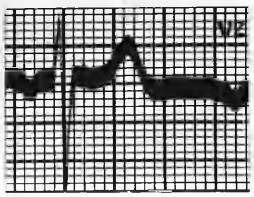
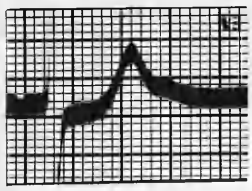
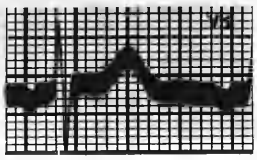
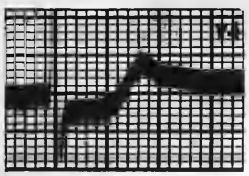
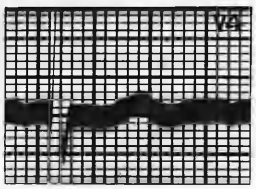
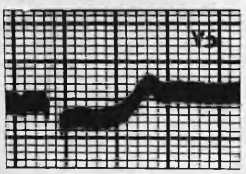
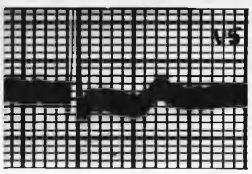
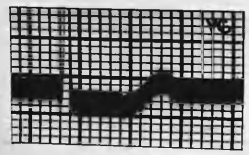
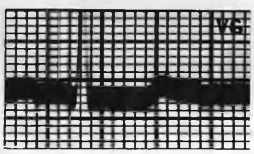
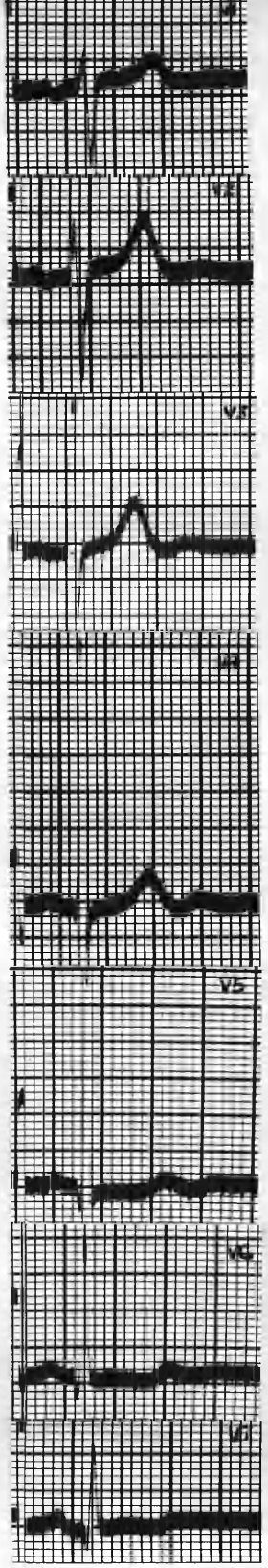



















CASE 41,
H.M. MALE, 51 YEARS.

1 DAY AFTER ONSET.

5 WEEKS AFTER ONSET.

7½ MONTHS AFTER ONSET.

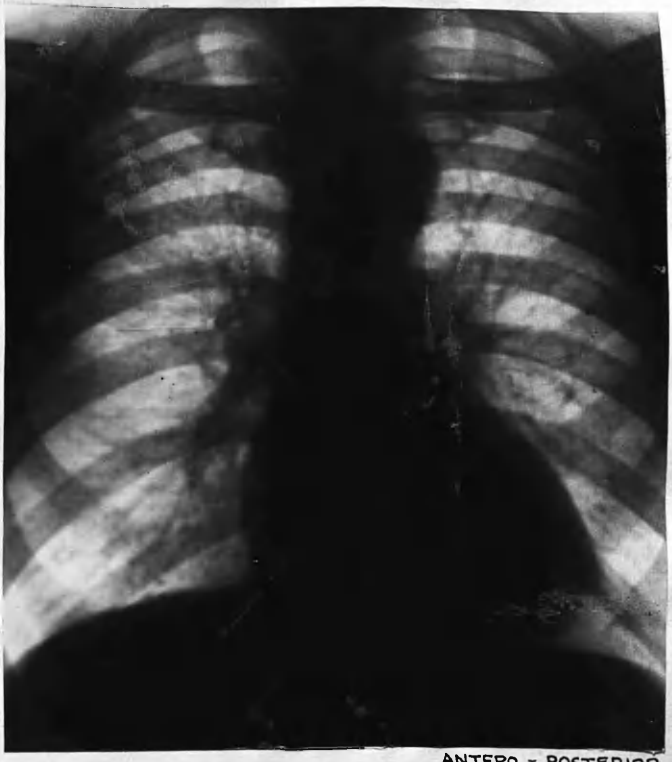














CASE 41,
H.M. MALE, 51 YEARS.

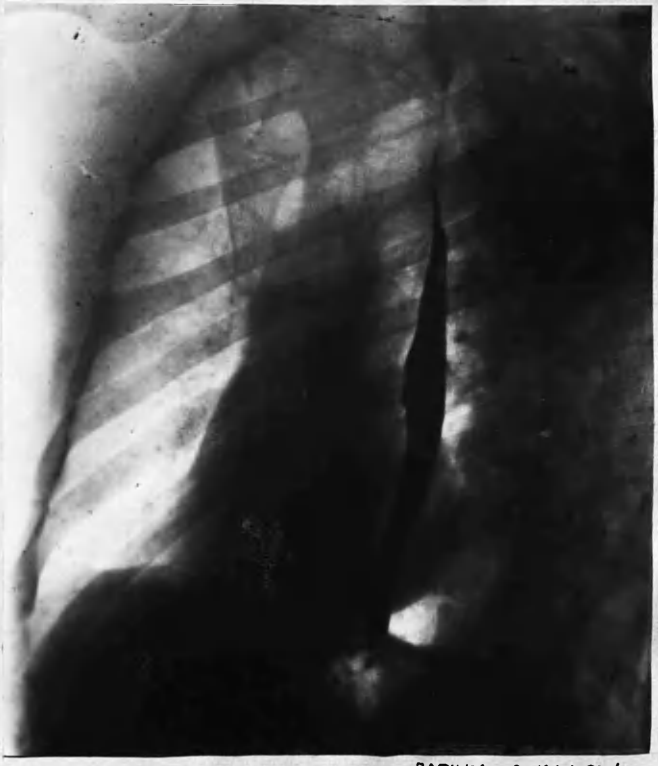
1 DAY AFTER ONSET.

5 WEEKS AFTER ONSET.

7½ MONTHS AFTER ONSET.



ANTERO - POSTERIOR



BARIUM SWALLOW

Case 42. A.F., male, 64 years.

This patient had had constrictive pain across the chest on exertion for three years and on one occasion he had been off work for ten weeks. Six days before admission to hospital, the pain was more intense and occurred while resting; it was accompanied by choking sensations, dyspnoea and flatulence. Cough was troublesome. Blood pressure was 120/90; cardiac sounds were soft and there were moist rales at both bases. An electrocardiogram one day after admission to hospital, i.e. one week after onset of illness shows the typical signs of recent infarction in lead VF, carried over also into leads II and III. Lead VL by its resemblance to the anterior rather than the lateral praecordial leads apparently reflects the potentials of the right side of the praecordium rather than the lateral wall of the left ventricle. There is severe depression of the ST segment in leads V3, V4 and V5 and slight depression in V6. Coupled with the tall R waves in V3 and V4 the ST depression is more likely to be due to the reciprocal effect of the posterior infarction than to subendocardial ischaemia of the anterior heart wall, especially since it is greatest in leads V3 and V4, i.e. opposite the infarction, and not in V5 and V6 which border on the region of infarction. Attention should be given to the direction of the initial phase of the QRS complex in leads V1, V2 and V3. In lead V1 there is a very small /

/ small primary R wave which is normal, followed, however, by abnormal broadening and splintering of the remainder of the complex. In leads V2 and V3 there is a small initial Q wave. This early negativity of the right ventricular cavity may be due to septal involvement, the initial negative potentials of the left ventricular cavity being transmitted through the infarct in the septum to the right ventricular cavity opposing the positive potentials developing therein as a result of activation of intact septal remnants.

During his second week in hospital respiratory symptoms were troublesome and signs of consolidation appeared at the right pulmonary base. Blood pressure fell further to 100/70. Two weeks after onset, an electrocardiogram shows typical sequential T changes evolving in the standard and unipolar limb leads. Reciprocal effects are disappearing from leads V3 and V4; the R waves have diminished considerably in height, the ST segment is no longer depressed and the T waves are increasing. However, the reduction in the R wave in the anterior praecordial leads had affected leads V1, V2 and V3 to a relatively greater extent than lead V4 in which lead the ST depression was greatest. This raises the possibility that another factor additional to the disappearance of reciprocal effects is responsible. In view of the respiratory complications it is probable that right ventricular strain was also present, causing further diminution in the height of the R wave in the anterior praecordial leads but the /

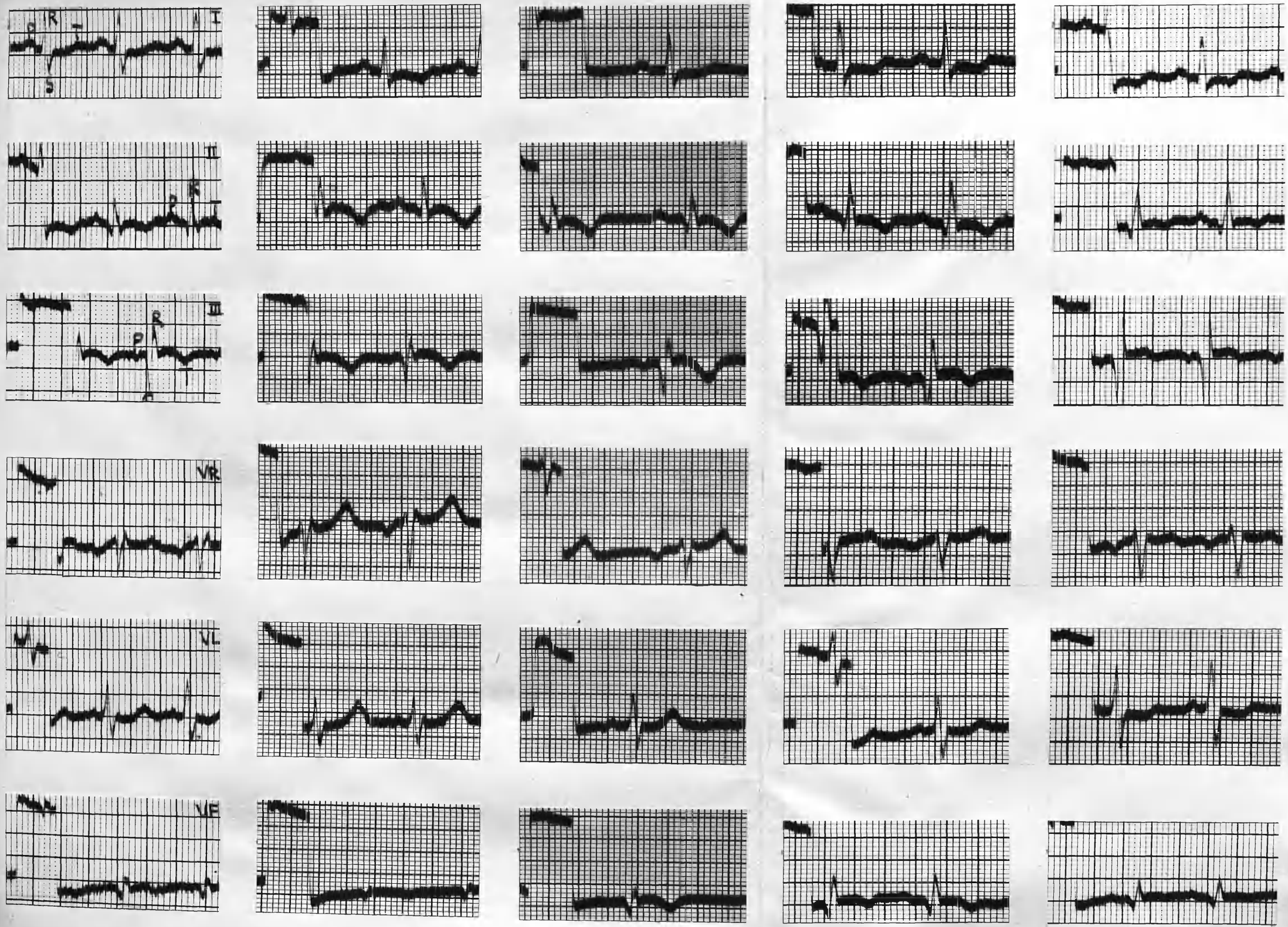
/ the reciprocal effects of posterior infarct on their T waves are severe enough to obliterate any direct effect of the right ventricular strain. It is typical that the tall T waves should occur in an outlying zone to the medial side of the lead where the ST depression is greatest. The Q wave previously noted in lead V2 has disappeared but persists in lead V3. While its presence does raise the possibility of patchy septal involvement, such a QRS pattern in leads V2 and V3 may be entirely explained by right ventricular dilatation which was evident clinically at this stage. The axillary leads still show some ST depression with shallow inverted T waves, which, in view of the fact that ST depression has disappeared from leads V2 and V3 where it was greatest, raises the possibility that in the axillary leads another cause is operating, probably subendocardial ischaemia.

Six weeks after onset, signs of posterior infarction persist in the standard and unipolar limb leads. The R wave is rising again in leads V2 and V3 but the Q wave of probable septal involvement is clearly seen in leads V2 and V3. The axillary leads still show signs of subendocardial ischaemia.

The patient left hospital after a stay of two and a half months. He was free of cardiac pain but exertional dyspnoea was readily induced. An electrocardiogram at this time shows the fixed pattern of posterior infarction in the standard and unipolar limb leads. The anterior praecordial /

/ praecordial leads show little change, whereas the ST-T formation of the axillary leads is now suggestive of left ventricular dilatation or hypertrophy. Lead VL now resembles lead V5 rather than the anterior praecordial leads suggesting that the left ventricular dilatation caused the heart to shift into a more horizontal position. The last electrocardiogram taken four months after onset shows a prominent Q with slurred R wave in lead VF indicating a residual conduction defect in the posterior cardiac wall. The axillary leads show no further change. The QS deflection of lead V1 may be a normal variant or may represent a fixed residue from patchy septal involvement. Lead V2 is normal, its Q wave having disappeared so that either the right ventricular dilatation has diminished or there has been some regression in the septal lesion.

This case illustrates the effects of multiple lesions: a long previous history of angina pectoris; an acute posterior infarction; subendocardial ischaemia of the lateral ventricular wall; right ventricular strain due to respiratory complications and the possibility of patchy septal involvement. Ultimately the signs indicate a localised conduction defect of the posterior wall with left ventricular dilatation or hypertrophy.



CASE 42, A.F., MALE, 64 YEARS.

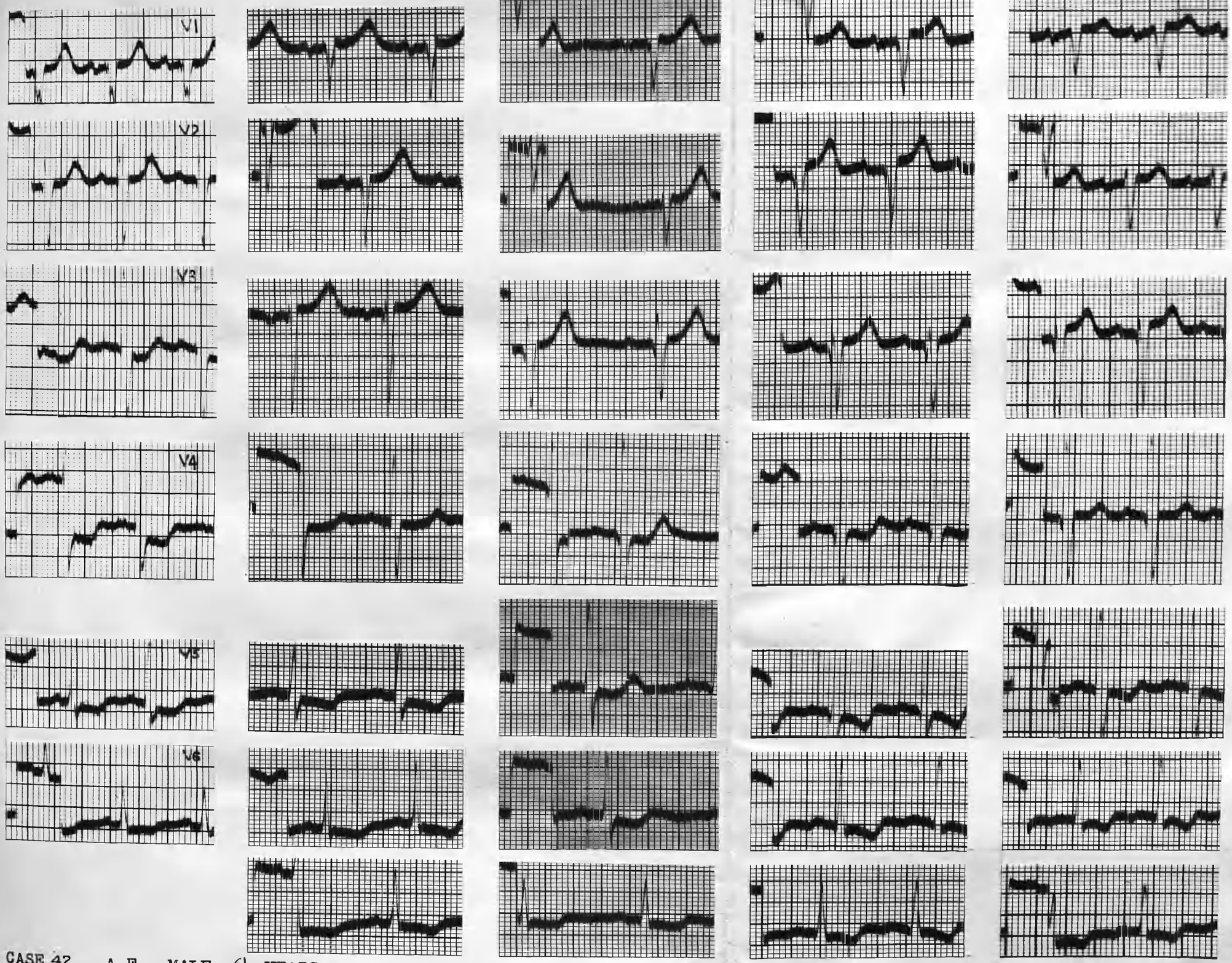
1 WEEK AFTER ONSET.

2 WEEKS AFTER ONSET.

6 WEEKS AFTER ONSET.

2½ MONTHS AFTER ONSET.

4 MONTHS AFTER ONSET.



CASE 42, A.F., MALE, 64 YEARS.
1 WEEK AFTER ONSET.

2 WEEKS AFTER ONSET.

6 WEEKS AFTER ONSET.

2½ MONTHS AFTER ONSET.

4 MONTHS AFTER ONSET.

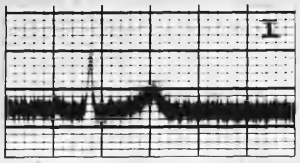
Case 43. J.R., male, 52 years.

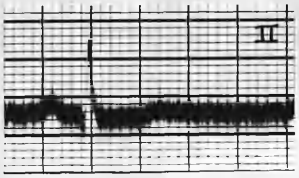
This patient had had angina of effort for two years preceding the attack of prolonged substernal pain which signalised the onset of coronary occlusion. When seen two months later at the out-patient department, B.P. was 130/82 and the cardiac sounds were soft. An electrocardiogram shows changes in lead VF strongly suggestive of infarction; there is a Q wave measuring 20-25% of the amplitude of the succeeding R wave, but only occupying 0.02 sec. in duration. There is slight depression of the ST segment and a shallow diphasic T wave. This pattern suggests an organising subendocardial infarct of the posterior wall. The praecordial leads are within normal limits.

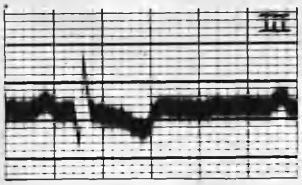
Patient was seen again fully one year later. He had resumed work as a teacher and felt well. B.P. was 120/60 and cardiac sounds were of medium intensity. An electrocardiogram shows little change in the standard limb leads but comparison with leads VL and VF, however, shows that the residual large Q wave of lead III is as much the reverse of the R wave in lead VL as it is the direct effect of the now tiny Q wave of lead VF - that is, in a semi--vertical heart, Q3 may be the result of initial positivity of the left arm. Both leads VR and VL show significant changes - there is now a deep S wave in lead VR and an S wave has appeared in lead VL. Similarly in the praecordial leads/

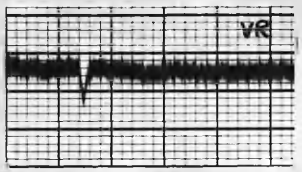
leads S has lengthened considerably in leads V1 through V4. These changes indicate increase in the negativity of the ventricular cavities. That it is the left ventricle that is mostly involved is seen by the corresponding increase in R in leads V5 through V7. Leads V6 and V7 show slight depression of the ST segment suggesting that there is actually a minor degree of left ventricular hypertrophy. In retrospect it is seen that the upright T in the original lead V1 was abnormal for this patient and that with the recovery of potentials mediated by the posterior wall and normally opposing the positive waves in leads over the right side of the praecordium, the T wave of lead V1 has reverted to an inverted form. The prominent R in lead VF along with the equiphasic RS complex in lead VL indicate that the heart is in the semi-vertical position.

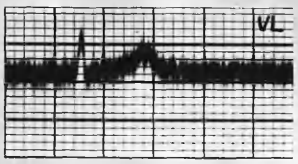
This case is an example of compensatory left ventricular changes subsequent to a well healed endocardial infarct of the posterior wall. Radiological examination shows a normal cardiac shadow.

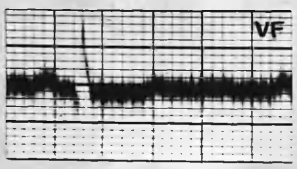




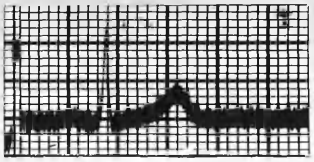


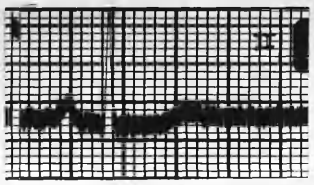


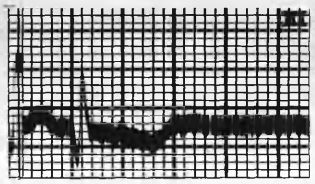


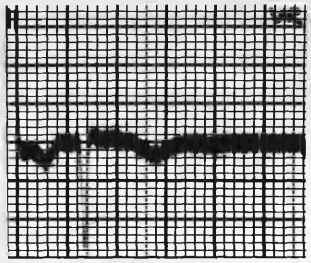


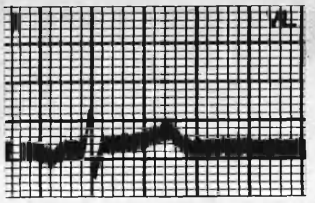
CASE 43,
J.R. MALE, 52 YEARS.
2 MONTHS
AFTER ONSET.

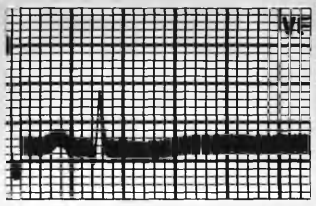




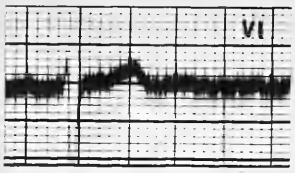


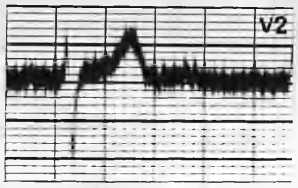


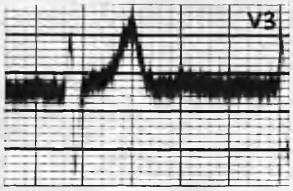


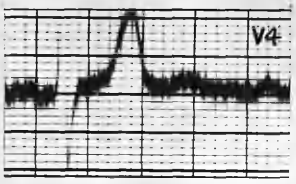


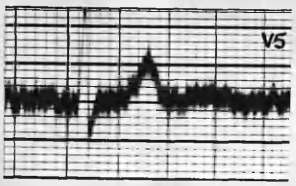
15 MONTHS
AFTER ONSET.

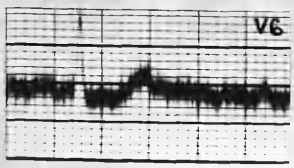




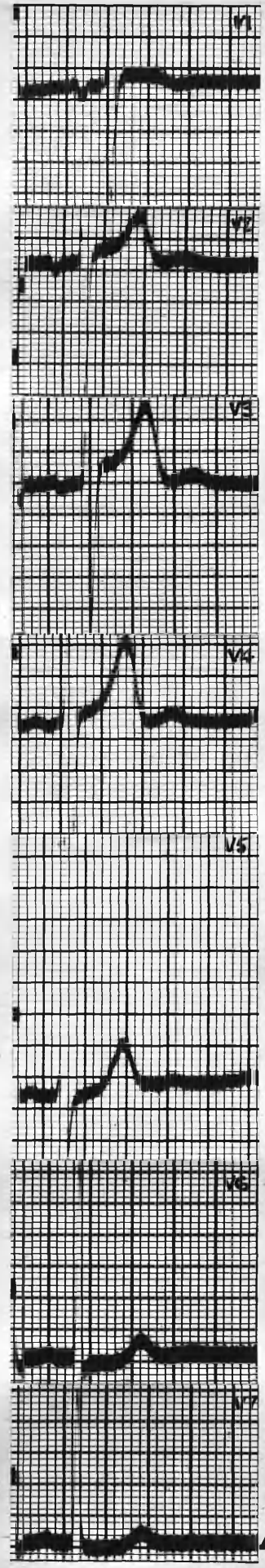




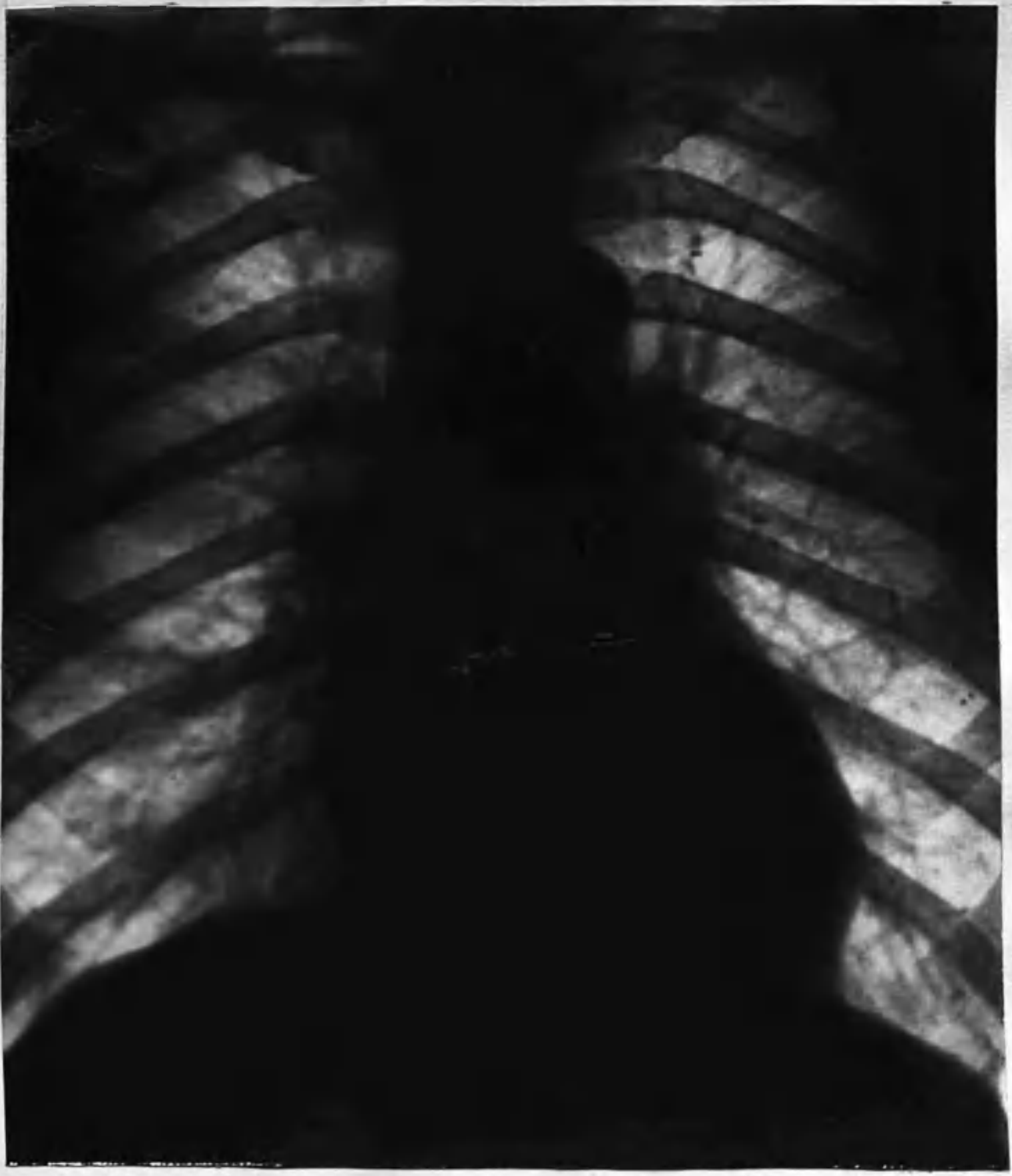




CASE 43,
J.R. MALE, 52 YEARS.
2 MONTHS
AFTER ONSET.



15 MONTHS
AFTER ONSET.



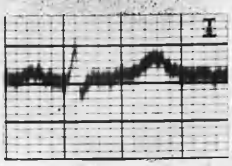
ANTERO - POSTERIOR

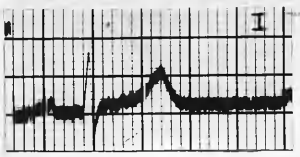
Case 44. D.P. male. 61 years.

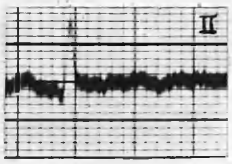
This patient aged 61 years had an attack of substernal pain lasting two days, seven and a half weeks previous to the first electrocardiogram. Lead VF shows a Q wave of 1 mm. or only 15 per cent of the succeeding R wave. The ascending limb of the R wave is slurred and slightly delayed; there is slight elevation of the ST segment and inversion of the T wave. These facts are suggestive, though not pathognomonic of posterior infarction, probably subendocardial. The signs in lead VF are carried into leads II and III. Praecordial lead V2 shows a very tall T wave commonly found in the anterior praecordial leads in posterior infarction. Lead V6 shows a small Q wave with slightly delayed R wave suggesting patchy extension of the posterior infarct into the lateral wall. Throughout the following year patient had occasional substernal pain at rest but he was ultimately well enough to return to light work.

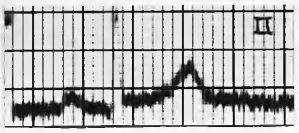
An electrocardiogram taken one year and eight months after, shows standard and unipolar limb leads which are normal for a vertical heart. The praecordial leads compared with the original tracing show an increase in depth of the S waves in leads VR and VL and in leads over

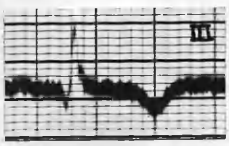
the right side of the praecordium along with heightened R waves and upright T waves over the left side of the praecordium. These signs indicate restoration of function of left ventricular muscle. The registration in the apical and axillary leads of QRST complexes which are manifestly left ventricular in origin, coupled with the sudden change from a right to a left ventricular pattern at the V3 - V4 position, indicates that the apex of the heart has been rotated forwards so that the lateral wall is subjacent to the electrodes in the V5 through V7 positions. There are no residual signs of infarction; it is noteworthy that the R wave of lead VF has not shared in the general increase in height although its Q wave has disappeared and its T wave is now upright. Radiologically there was no cardiac abnormality; the thorax was Kyphotic which may have influenced the position of the heart.

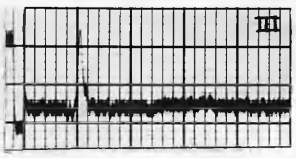


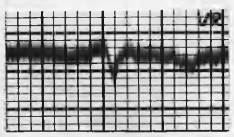


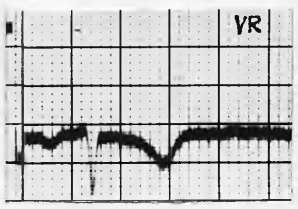


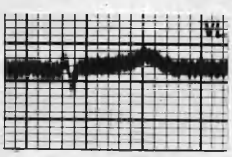


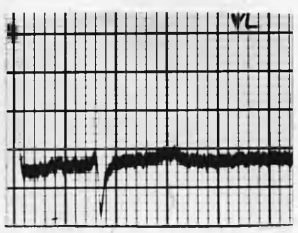


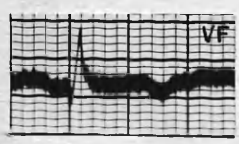


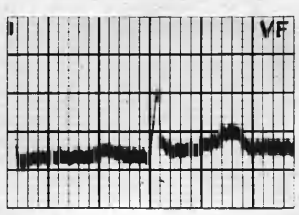




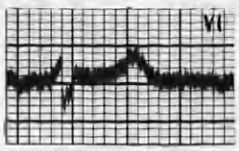


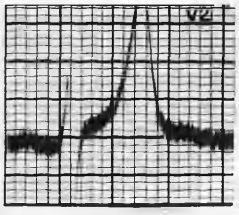


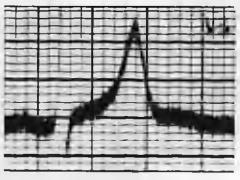


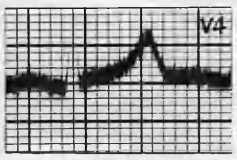


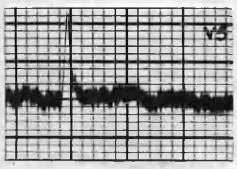
CASE. 44, D.P.MALE 61 YEARS.
7½ WEEKS 1 YEAR 8 MONTHS
AFTER ONSET. AFTER ONSET.

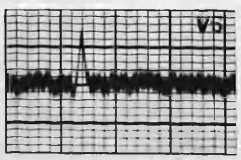




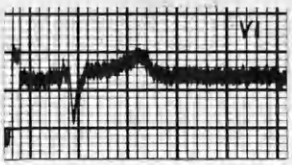


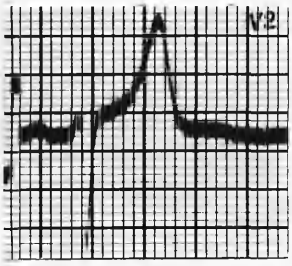


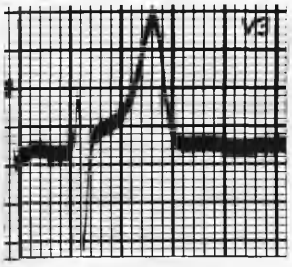


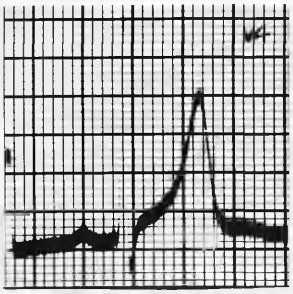


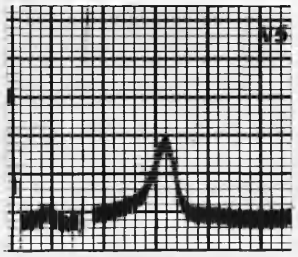
CASE. 44,
D.P. MALE, 61 YEARS.
7½ WEEKS
AFTER ONSET.

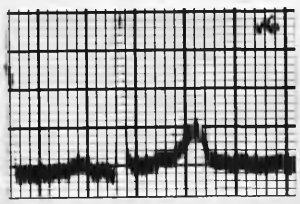


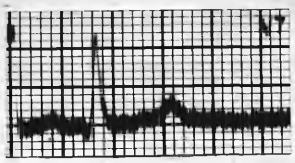




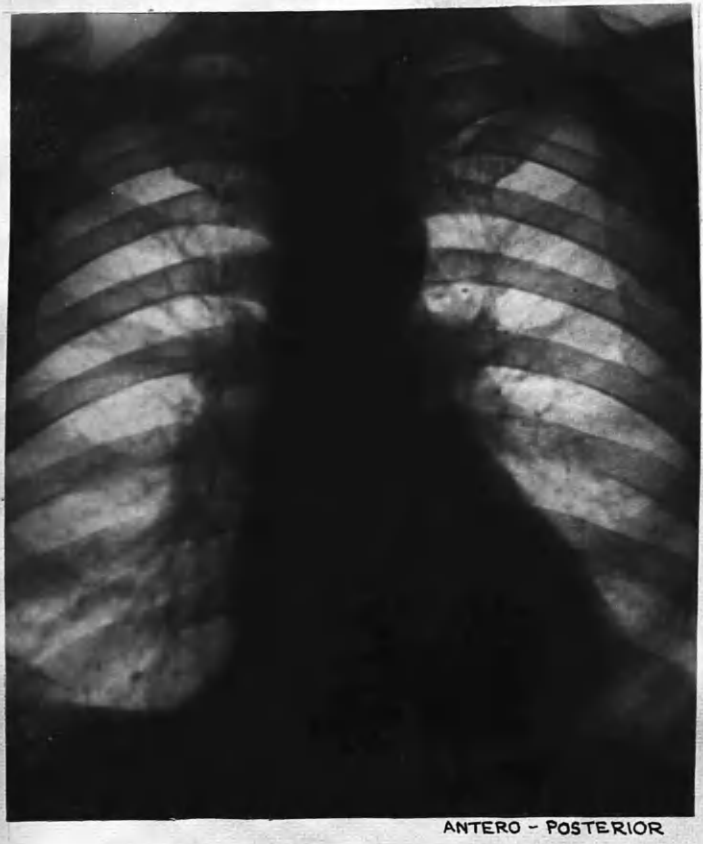




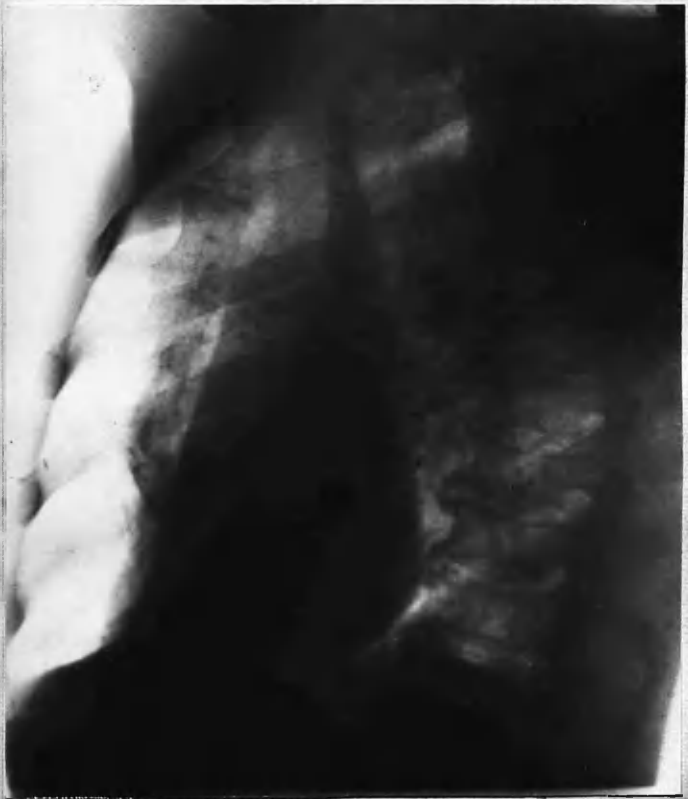




1 YEAR 8 MONTHS
AFTER ONSET.



ANTERO - POSTERIOR



LEFT ANTERIOR OBLIQUE

Case 45. B.A.M., 50 years, male.

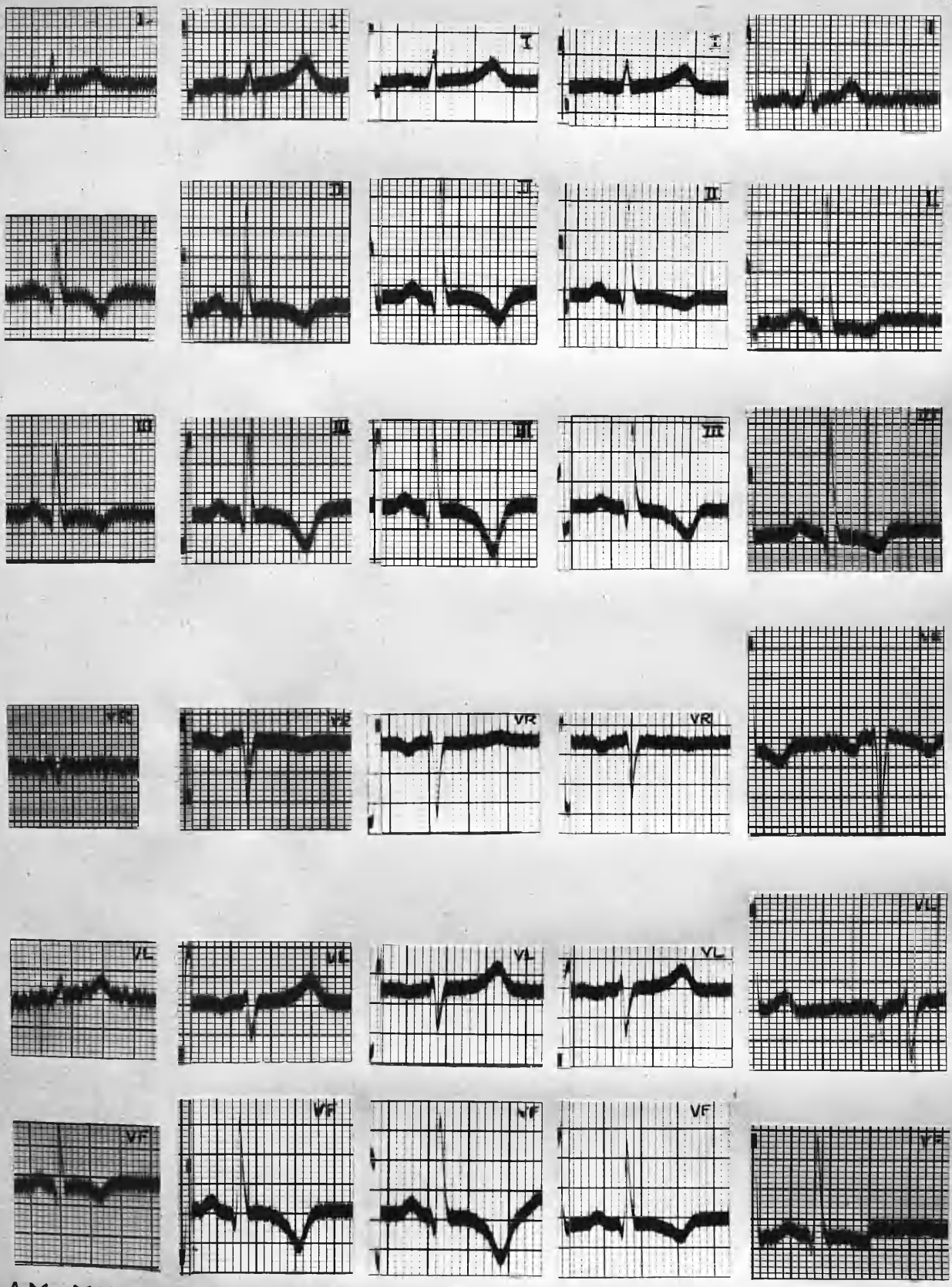
This patient gave a history of severe substernal pain with collapse one week previous to the first electrocardiogram. Lead VF shows a small Q wave occupying 0.02 sec. and measuring not more than 20% of the succeeding R wave. Thus neither the Goldberger not the Myers criteria are fulfilled. The dome-like ST and inverted T suggest an organising lesion. The findings in lead VF are closely copied in leads II and III. Although analysis of this electrocardiogram fails to establish the diagnosis of posterior infarction, consideration of the changes that followed leaves no doubt that a sub-endocardial posterior infarct did exist. Lead VF and also leads II and III show in the ensuing weeks flattening of ST and the development of a large cove-plane T wave. These changes are associated with tall spike-like T waves in praecordial leads V1 through V4, without exaggeration of the R waves, indicating that these leads are reciprocating the electric potentials of the zone of ischaemia around the infarction. Lead V6 shows variable T waves, flat or inverted, more likely to be due to an outlying zone of ischaemia in the lateral wall of the left ventricle than to any pre-existing left ventricular hypertrophy.

There are also significant changes in the S waves. As early as four weeks after onset, S re-appears as a moderate or large deflection in leads VR and VL. In the praecordial leads it increases considerably in length in leads V1 through V3, but up to five weeks after onset, there is still no S wave in leads V4 and V5, the ST segment rising abruptly from the descending limb of R. At two months, however, it has re-appeared in lead V4.

This patient made an excellent recovery and when seen again one year after onset had resumed work as a clerk and was free of symptoms. An electrocardiogram at this date shows a QRS pattern in the standard and unipolar limb leads commonly found in vertical hearts. Radiologically the heart is also vertical. The contour of the T waves in leads VF, II and III has also changed and although they are the residue of cove-plane T waves, their present shallow inverted or diphasic form is not that of a fixed posterior infarct. Furthermore, a shallow inverted T wave in these leads may occur as a normal variant in a vertical heart with forward rotation of the apex. The praecordial leads show a further late increase of S in leads over the right side of the praecordium and its re-appearance now in lead V5. These S changes indicate a full return of left ventricular electrical potentials

transmitted through the septum and right ventricle as these structures become depolarised. By one year after onset, the ST segment has risen to its normal level above the base line and the T waves are shorter, the decrease in height starting in lead V1 first. It is noteworthy that the R waves, while tall in lead V4, are not abnormally so. The fact that the main QRS changes are borne by the S waves in leads over the right side of the praecordium is to be correlated to the vertical position of the heart which militates against the accessibility of the left ventricle to the praecordial electrode in leads over the left side of the praecordium. Lead V7 shows a small Q wave followed by a slurred upstroke of small voltage pointing to a conduction defect in the postero-lateral wall of the left ventricle in the proximity of the infarction. This case is an example of the very satisfactory healing of a subendocardial patchy or laminar infarction of the posterior wall of the left ventricle. The failure of the original lead VF to show signs diagnostic of posterior infarction may be due to the fact that when the heart is in an intermediate to vertical position, the infarct may not be in a position to influence directly the potentials of the left leg.

Especially is this the case if the infarct is in the basal third of the free wall, but it is less likely if the apical third is involved whereas diagnostic failures in lead VF are rare if the middle third is involved.



No45, B.A.M., MALE, 50 YEARS.

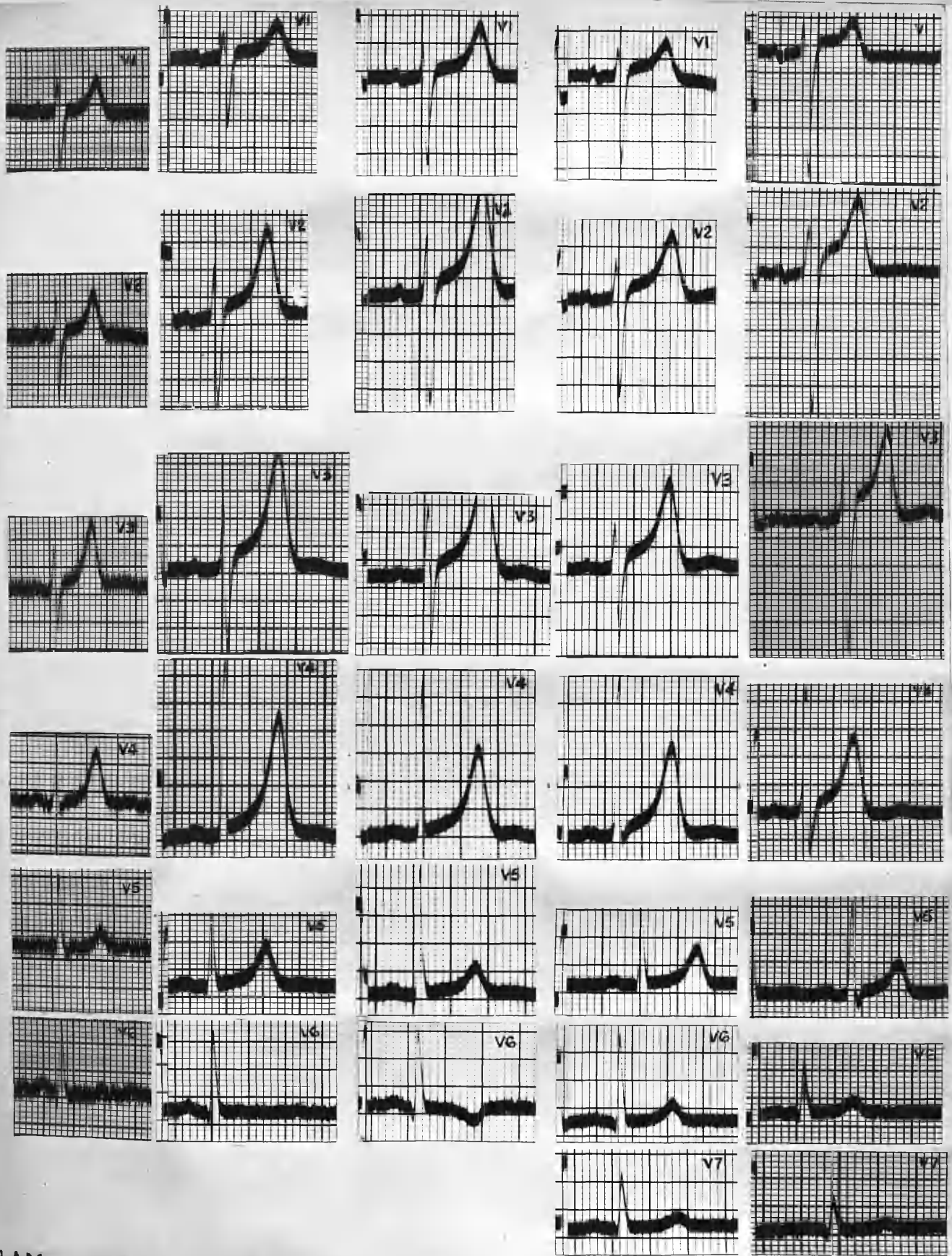
1 WEEK AFTER ONSET.

4 WEEKS AFTER ONSET.

5 WEEKS AFTER ONSET.

2 MONTHS AFTER ONSET.

1 YEAR AFTER ONSET.



45, B.A.M., MALE, 50 YEARS.

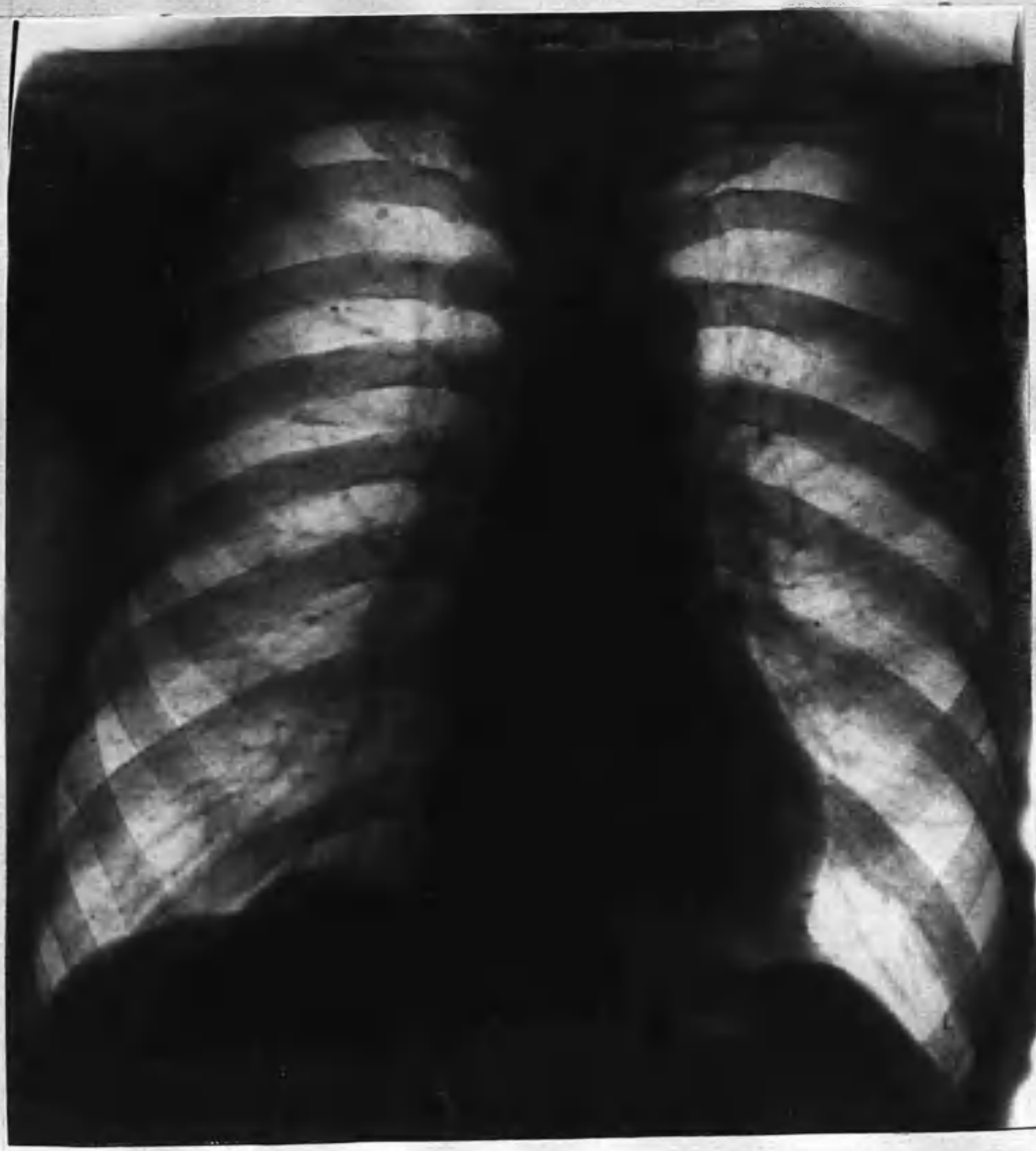
1 WEEK AFTER ONSET.

4 WEEKS AFTER ONSET.

5 WEEKS AFTER ONSET.

2 MONTHS AFTER ONSET.

1 YEAR AFTER ONSET.



ANTERO - POSTERIOR

Case 46. A.C., male, 54 years.

This patient had sudden severe pain across the anterior chest wall, which came on while at work. He attended the out-patient department five weeks later, when the first electrocardiogram was taken. Lead VF presents a small Q consuming 0.02 sec. from onset to nadir (or slightly less than 0.04 sec. from onset to where its upstroke again reaches the base line); in amplitude it measures 25% of the succeeding R wave. These features do not entirely fulfil the Goldberger criteria, (the Q wave in lead VF should have a duration of 0.04 sec. from onset to return to base line and amplitude of 60% of the succeeding R wave). It has been pointed out by Myers et al that Goldberger's criteria eliminate some cases of posterior infarction. These workers consider that in lead VF, QR complexes of 0.5 millivolt or more are diagnostic of posterior infarction when the Q wave measures 0.03 sec. or more from onset to nadir and exceeds 25% of the amplitude of the associated R wave. If one of those two requirements are met, they are classed as borderline to strongly suggestive. Furthermore, they maintain that a QR complex of 0.5 millivolt or more in lead VF characterised by a distinct Q followed by a prolonged or coarsely slurred upstroke is considered abnormal, even though the duration of the Q wave and the Q/R ratio do not meet the foregoing minimal requirements. It /

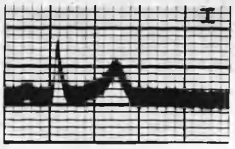
/ It will be seen that lead VF of the case under discussion presents sufficient evidence to justify the diagnosis of posterior infarction. Furthermore, since the Q wave reflects the initial negativity of the left ventricular cavity, the slurring and delay in the ascending limb of the R wave are to be ascribed to a conduction defect of the free posterior wall of the left ventricle. Such a QR pattern indicates patchy infarction, islands of intact myocardium surviving amidst the organising tissue. The dome-shaped ST segment and inverted T wave are also suggestive of a recent infarction in process of organisation. The findings in lead VF are carried over into lead III, but lead II is only slightly affected.

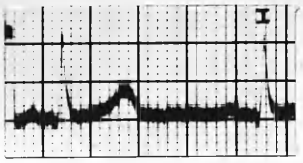
The praecordial leads show persistence at five weeks of high spike-like T waves in leads V3 and V4 and the abrupt manner in which the ST segment leaves the descending limb of R before it reaches the base line in lead V4 is a common finding at this stage.

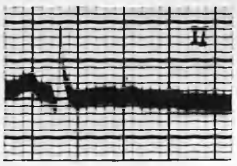
This patient made an excellent recovery and when seen two years later, was working as a goods-checker. He was free of symptoms. An electrocardiogram shows a normal lead VF, the tall R being typical of a vertical or semi-vertical heart. Lead VL shows a multiphasic QRS of low voltage, which is a normal finding in semi-vertical positions. The re-appearance of the S wave in this lead is consistent with an increase in the negative potentials of /

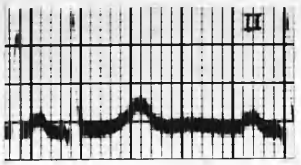
/ of the left ventricular cavity. Recovery of left ventricular function is also recorded in the increased S wave in lead V1 through V3, and a small S has also returned to lead V5 eliminating the abrupt origin of ST from the descending limb of R as previously noted. The wave R has also increased in lead V4 through V7. It is possible that these changes indicate a minor degree of left ventricular hypertrophy. The T waves have returned to normal and in retrospect, it is seen that the previously upright T wave in lead V1 and the rather large T wave in lead V2 are both abnormally positive for this patient and attributable to the reciprocal effect of the infarction. Radiologically there was slight enlargement of the left ventricle.

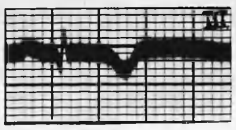
This case is an illustration of a well healed posterior infarct. It was probably patchy or laminar and partly endocardial, not transmural, in the first instance. Slight left ventricular hypertrophy has occurred as a compensatory phenomenon.

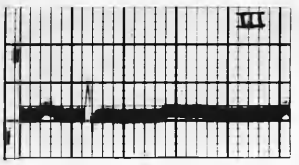


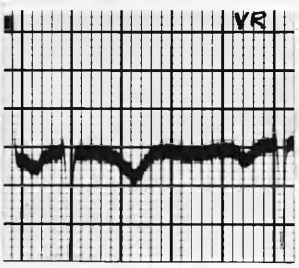


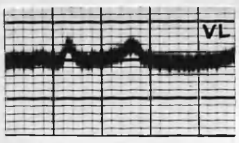


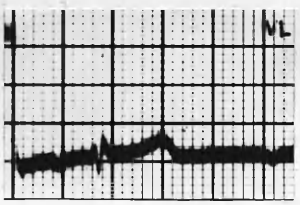


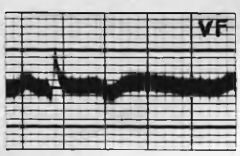


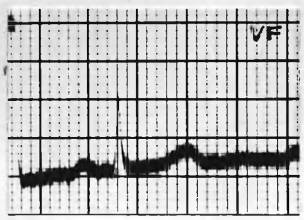








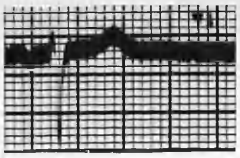


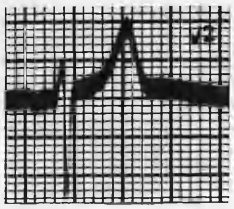


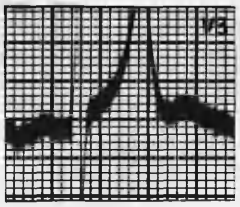
CASE. 46, A.C. MALE, 54 YEARS.

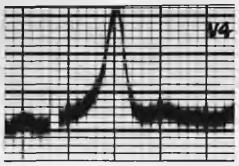
5 WEEKS AFTER ONSET.

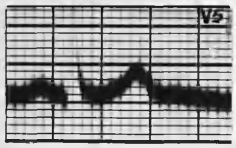
2 YEARS AFTER ONSET.

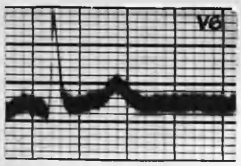




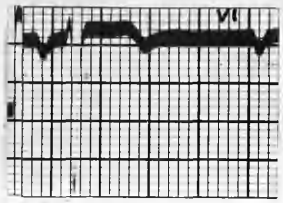


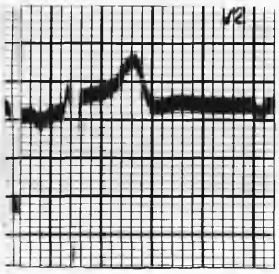


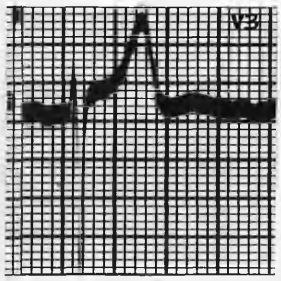


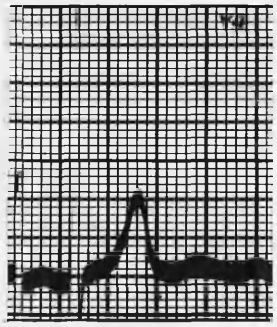


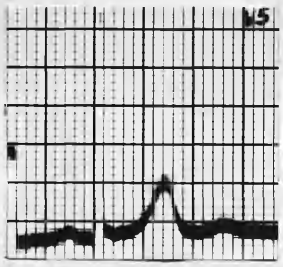
CASE. 46,
A.C. MALE, 54 YEARS.
5 WEEKS
AFTER ONSET.

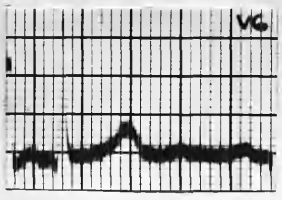


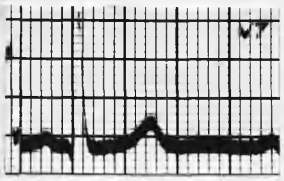




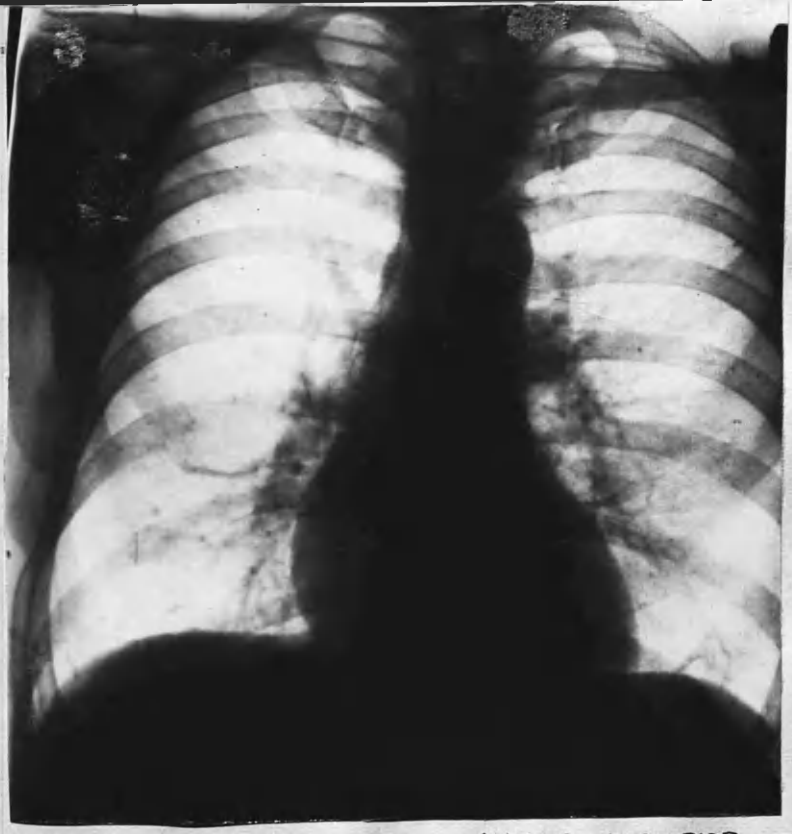




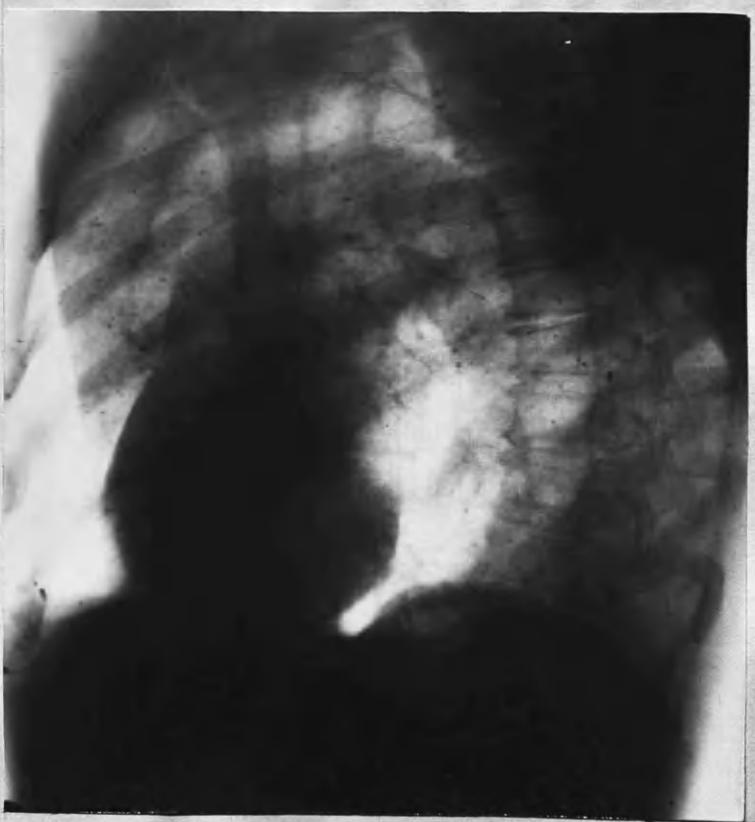




2 YEARS
AFTER ONSET.



ANTERO - POSTERIOR



LEFT ANTERIOR OBLIQUE

Case 47. R.C., male, 52 years.

This patient was admitted to hospital because of substernal pain recurring throughout two or three days. On admission there was no evidence of shock. Blood pressure was 130/80. There was no enlargement of the heart and the cardiac sounds were pure. There was no further chest pain and recovery was satisfactory.

An electrocardiogram taken five days after onset shows the typical picture of posterior infarction in the standard limb leads. There is no Q wave in lead I. The Q waves of leads II and III are very conspicuous; their R waves are relatively small; there is upward convexity of the RS-T segments and prominent inverted T waves. Lead VF is also typical; its width of 0.04 sec. should be noted. Lead VL shows a rapidly rising R wave at the termination of which is a minute SR' deflection. This is followed by an RS-T segment convex downwards and a pointed symmetrical upright T wave; in all particulars this pattern is typical of the left arm lead when it apposes a posterior infarction in an intermediate to horizontal heart. Lead VR shows a pattern derived from the anterior cardiac wall and resembles closely lead V1 at least in its QRS formation. Its normal r wave does not produce a Q wave in standard limb lead I because the positivity of the left arm is so immediate. The RS-T segment of lead VR is typical of that lead when in apposition to anterior or posterior infarction, viz. there is slight downward curving of the RS-T segment

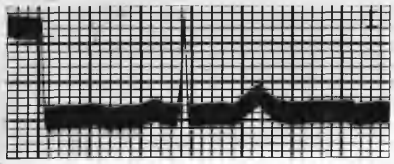
and/

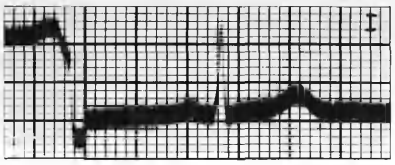
/and a prominent upright T wave.

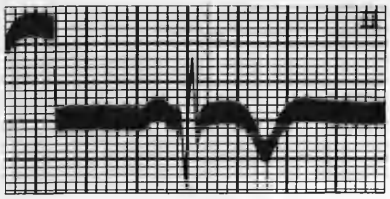
The praecordial leads show decrease in the height of the R wave in lead V3 compared to lead V4 - a sign suggestive of partial involvement of the anterior wall; in addition the large symmetrical downward T waves of leads V4 and V5 denote the zone of ischaemia around an anterior infarction. In view of the similarity of lead VR to leads V1 and V2 and to the transition zone being at lead V4 and not V3 it is probable that there was forward rotation of the apex combined with some clockwise rotation of the heart itself around its own long axis. The former movement would encourage the forward edge of an infarction of the apical region of the posterior wall to come into relation-ship with leads V3, V4 and V5. Probably there was also some actual involvement of the anterior part of the apex by the infarction.

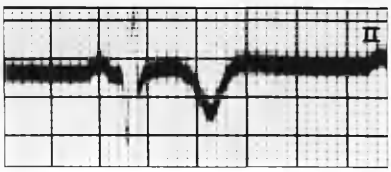
An electrocardiogram a week later shows little change: the R wave of lead V3 is larger again and the T wave of lead V5 much less inverted - both favourable signs.

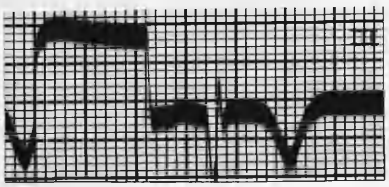
This case is noteworthy in so far as the standard limb leads, which show the classical signs of posterior infarction, give no indication of the anterior extension.

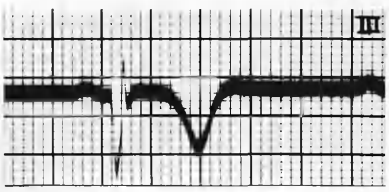


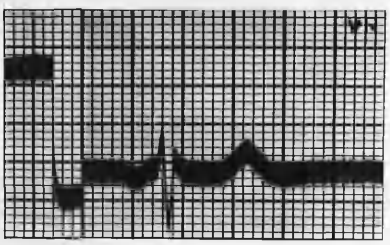


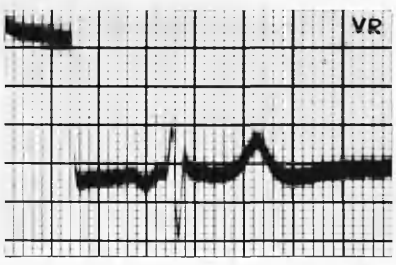


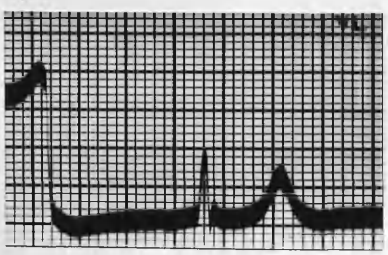


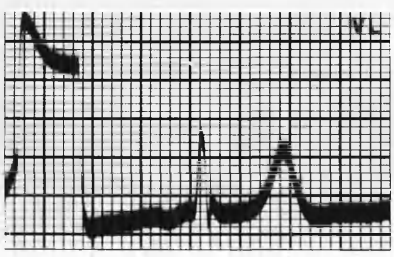


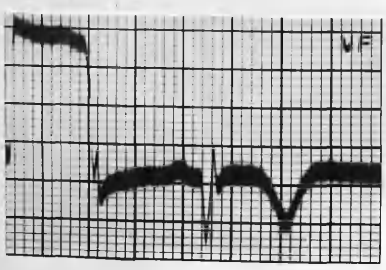


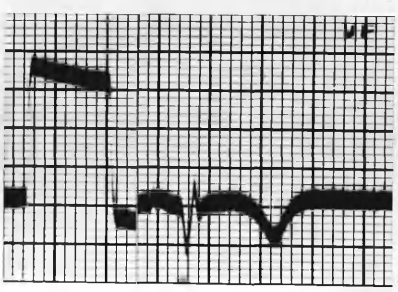








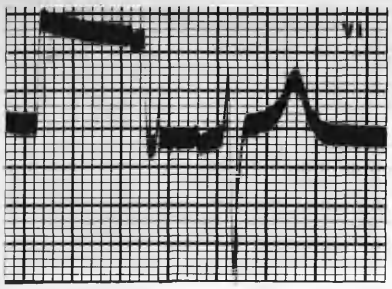


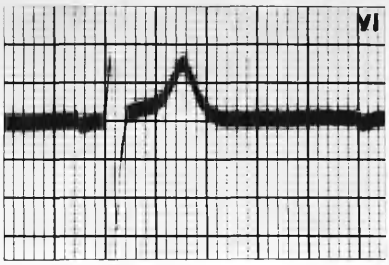


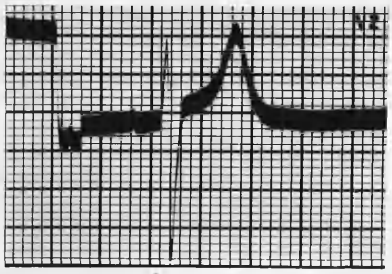
CASE 47, R.C., MALE 52 YEARS.

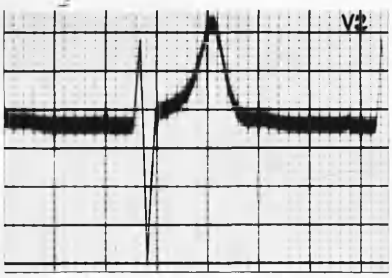
5 DAYS
AFTER ONSET.

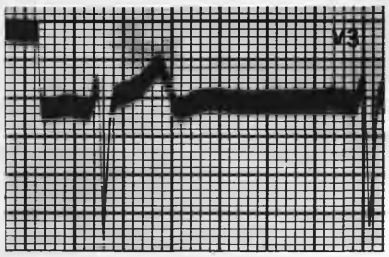
11 DAYS
AFTER ONSET.

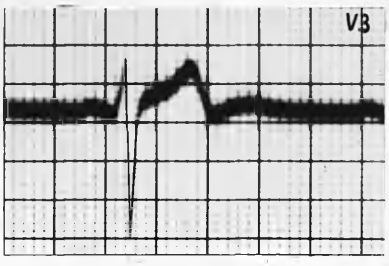


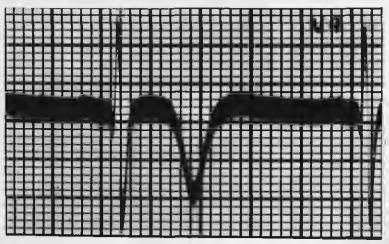


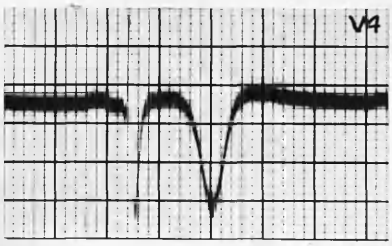


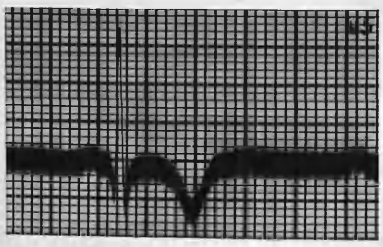


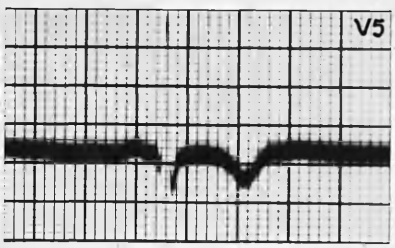


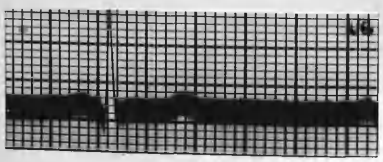


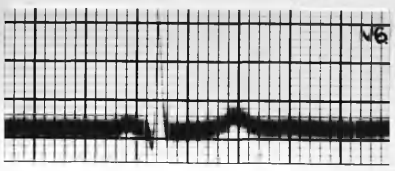












CASE 47, R.C., MALE 52 YEARS.

5 DAYS
AFTER ONSET.

11 DAYS
AFTER ONSET.

Case 48. W.S. Male, 54 years.

This patient, a laundry-worker, was seized with severe epigastric pain while at work. It radiated into the left side of the chest and was accompanied by nausea and sweating. On his return home the pain diminished after an injection of morphine. He remained in bed for a fortnight at home after which admission to hospital was arranged. Blood pressure was 130/85. The apex-beat was not palpable; there was no clinical enlargement of the heart; the cardiac sounds were of very poor quality.

The first 12-lead electrocardiogram was obtained two weeks after onset of illness. It shows the typical signs of an organising posterior infarction in standard limb leads 2 and 3. In addition, lead 1 shows flattening or very slight upward convexity of the ST-T segment; there is right axial deviation and slight broadening of the QRS deflection of leads 2 and 3. Lead VF confirms the presence of an infarction in the posterior wall; the height of the QRS deflection just reaches 0.5 millivolt; the Q/R ratio is about $\frac{1}{2}$; the ST segment

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/ shows a prominent upward convexity and the T wave is inverted. Lead VR with its QR deflection and sharply inverted P wave suggests a derivation from the back wall of the heart but the large symmetrical T wave is undoubtedly a reciprocal effect from the endocardial surface of the infarct.

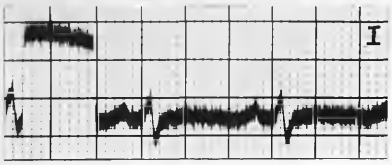
Praecordial leads V2, V3 and V4 have relatively small R and deep S waves indicating right ventrical origin. The transitional zone is between leads V5 and V6. Furthermore, lead VL shows an RS pattern. These signs together are strongly suggestive of a vertical or semivertical electrical position. This position partially accounts for the small size of the R wave of lead V6, but in view of the delay which it shows in attaining its peak and of the lateness of its equivalent in lead V5, where it forms a small R wave, there is probably some patchy or intramural involvement of the lateral wall of the left ventricle. This deduction is strongly supported by the RS-T formation - a low or iso-electric RS-T junction, a convexly upward RS-T segment and sharp inversion of the T wave -

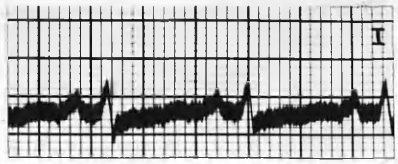
/

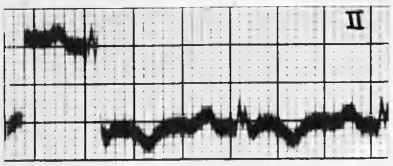
/ to be seen in leads V5 and V6. Thus the infarction can be localised to the posterior wall with probable intramural extension into the lateral wall.

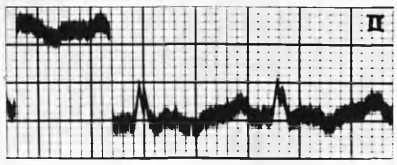
Two weeks later, i.e., four weeks from onset, there is little change in the standard limb leads.

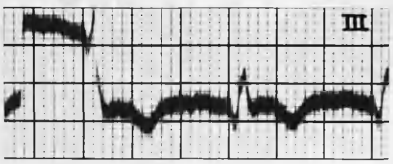
There is however, an increase in the R wave of the axillary and apical leads suggesting increased functional activity of the left ventricle, but the RS-T segment is virtually unaltered.

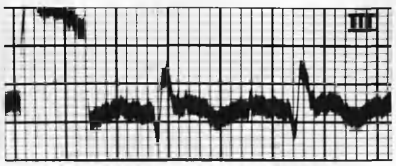


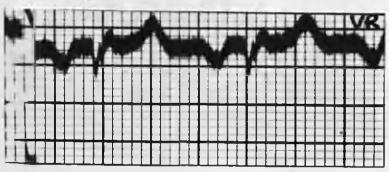


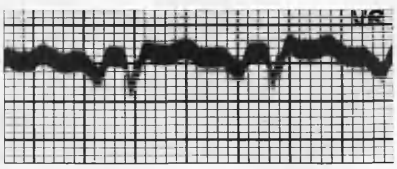


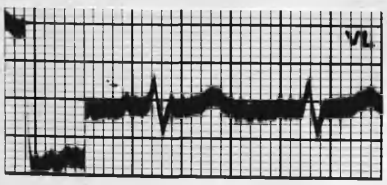


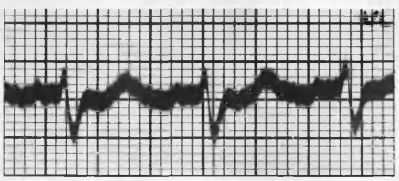


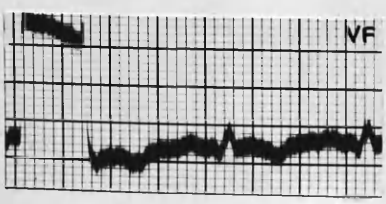


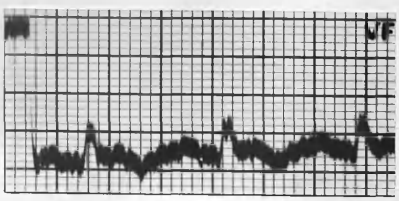








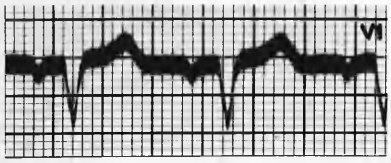


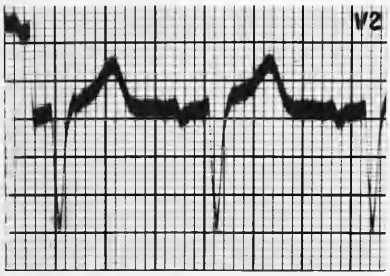


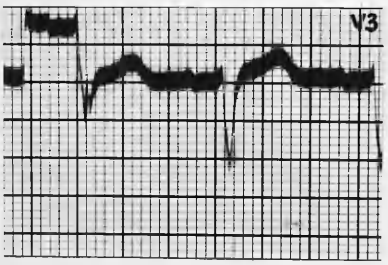
CASE 48, W.S., MALE 54 YEARS.

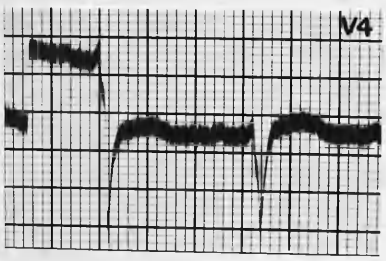
2 WEEKS
AFTER ONSET.

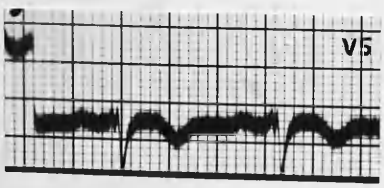
4 WEEKS
AFTER ONSET.

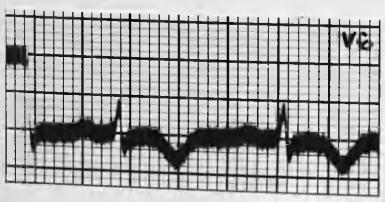


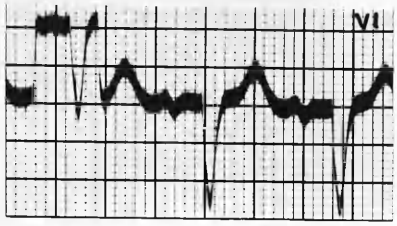


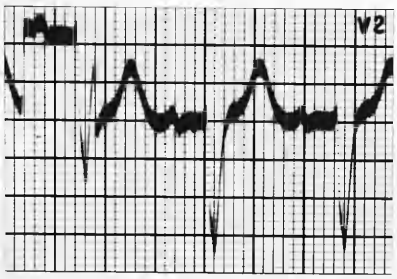


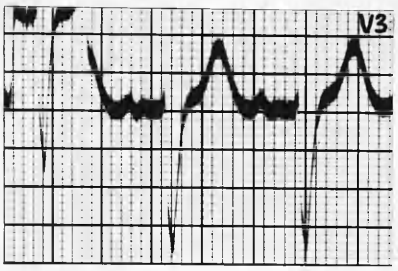


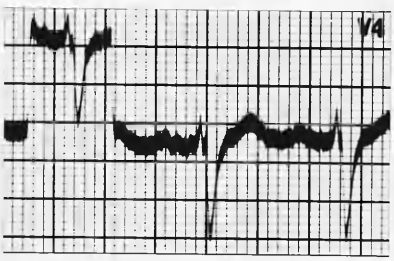


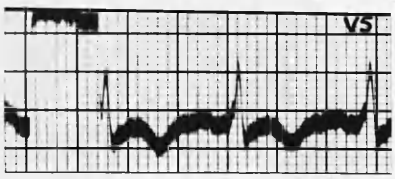


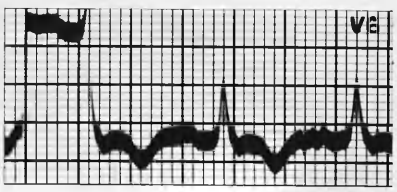












CASE 48, W.S., MALE 54 YEARS.

2 WEEKS
AFTER ONSET.

4 WEEKS
AFTER ONSET.

Case 49. H.R. Male, 60 years.

This patient, a dock labourer, was admitted to hospital two days after the occurrence of an aching pain in the praecordial area. Its onset was accompanied by faintness and sweating. After half an hour or so, the pain radiated into the left arm and across the chest to the right axilla. It was ultimately relieved by morphine. For the previous four to five months the patient had had breathlessness on unusual exertion but no chest pain.

Patient was a heavily built man. There was slight cyanosis of the lips and fingers. Blood pressure was 160/120. The cardiac sounds were poor and pericardial friction was audible. An electrocardiogram at this date, Viz. two days after onset shows the classical signs of recent posterior infarction in standard limb leads 2 and 3. Of the unipolar limb leads the tall R wave of lead VL suggests that the electrical position of the heart is horizontal to intermediate. The former position is the more likely in view of the association /

/ with the deep QS deflection of lead VF which is a common finding in the horizontal position. Either it may represent a normal variant or it may be a manifestation of a septal infarct which serves as a window through which the negative potentials of the left ventricular cavity are transmitted to the left thigh. The elevation of the RS-T junction and the plateau-like RS-T segment are strongly in favour of infarction. The depression of the RS-T segment in lead VL is a reciprocal effect from the infarct.

Lead V1 is somewhat similar to lead VF insofar as it presents a deep QS deflection, elevation of the RS-T junction and straightening of the RS-T segment. These findings are very suggestive of infarction of the interventricular septum since lead V1 is in much the same relation to the septum and left ventricle as is lead VF. Although more remote, lead VR also faces the right side of the septum; hence the upward convexity of the RS-T segment derived from the infarcted portion. The possibility that these RS-T signs are due to pericarditis, which was present clinically, must be considered.

/

/ However their limitation to a single praecordial lead is against their derivation from pericarditis. The other anterior praecordial leads show the reciprocal effects of posterior infarction, Viz., unduly tall R waves and large upright T waves.

During his first week in hospital blood pressure/ gradually fell to 114/84 about which level it remianed throughout his hospital stay of two months. Pericardial friction disappeared within two or three days. Radiological examination of the heart showed some generalised enlargement of the cardiac shadow with prominent vascular markings in the lung fields.

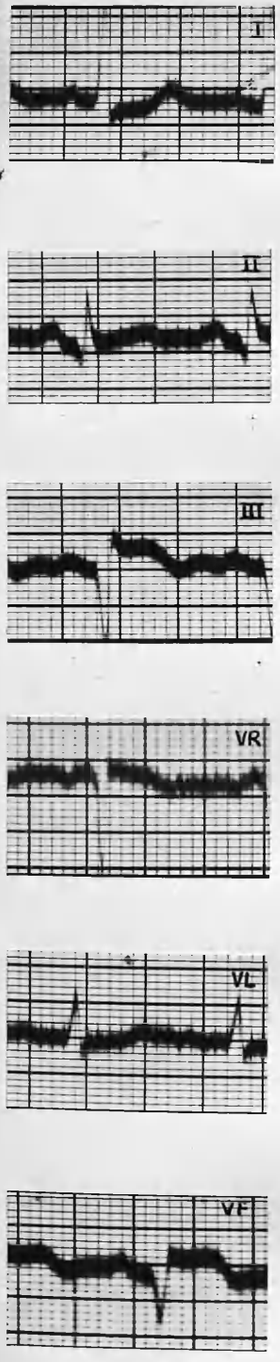
Tracings taken five days after onset show less elevation of the RS-T junction in the leads which formerly displayed it. By twelve days after onset the RS-T junction and segment are approaching the iso-electric level in lead VF but some elevation still remains in lead V1 and there is still slight reciprocal ST depression in lead VL and hence in standard limb lead 1. In the other praecordial leads, the T waves are much less conspicuous than formerly, partly because the exaggeration reciprocal to posterior infarct has disappeared and

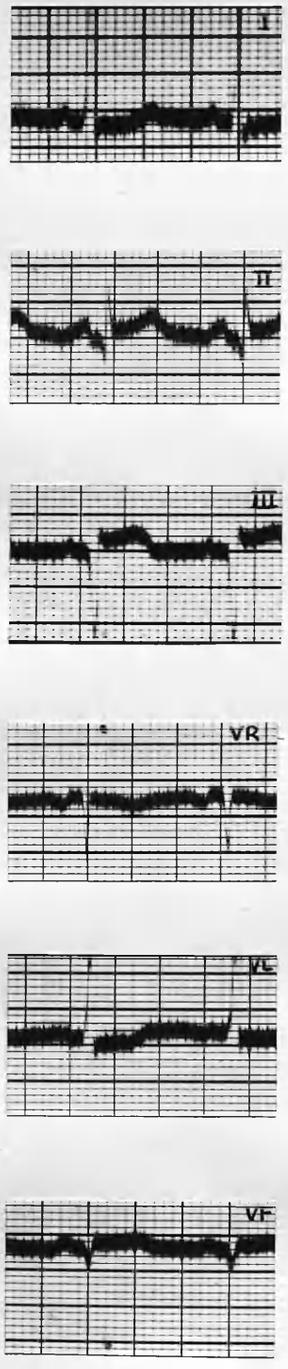
/

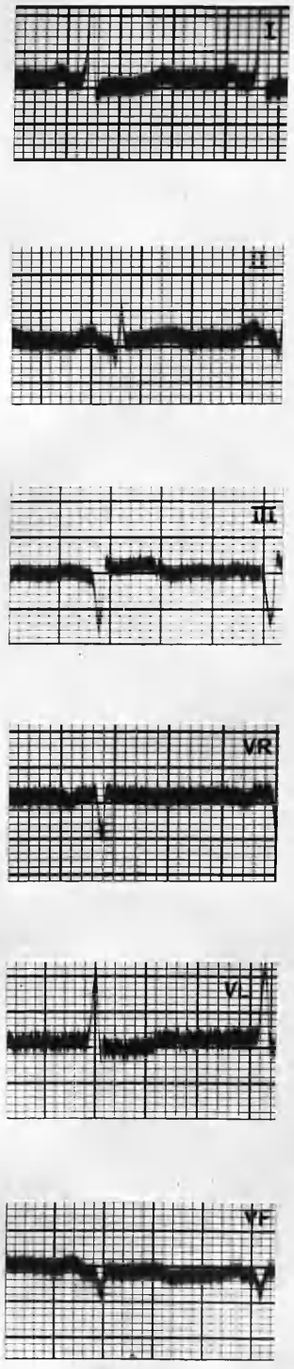
/ and partly as a result of pericarditis. In lead V2 the R wave has also diminished in height due to cessation of reciprocal augmentation.

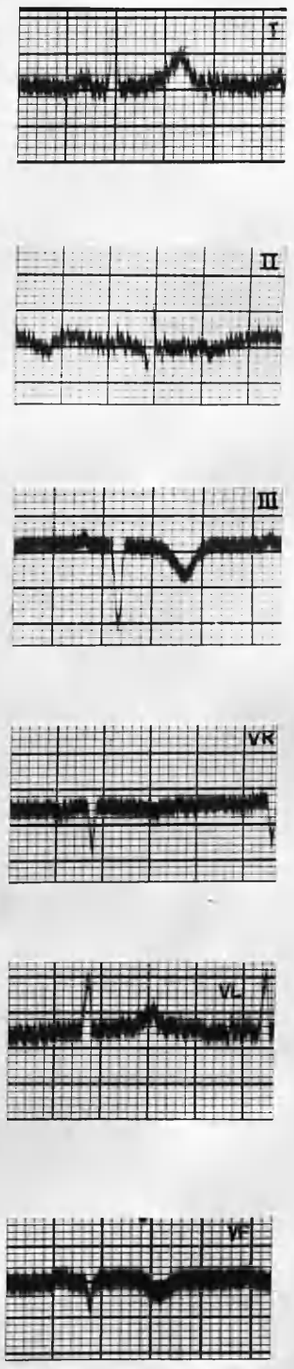
The last two electrocardiograms taken at four and five weeks after onset show the evolution of the coronary T waves in unipolar leads VF, V1 and in standard limb leads 2 and 3.

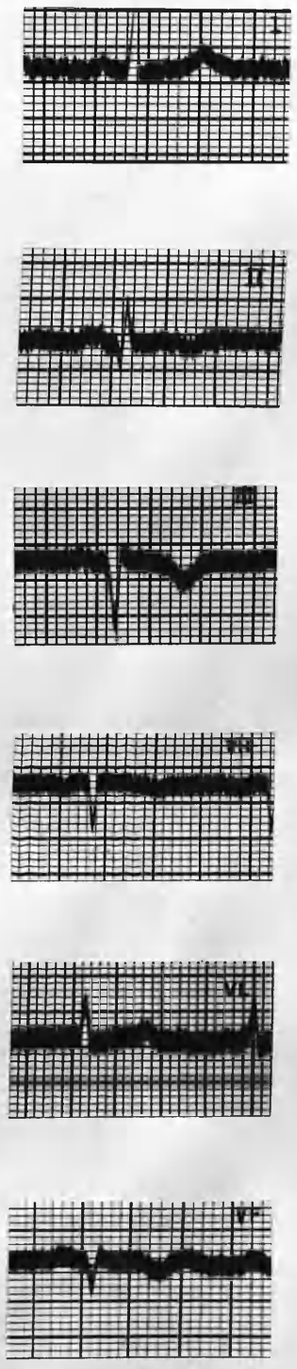
This case presents the sequential changes of posteroseptal infarction.











CASE 49, H.R., MALE 60 YEARS.

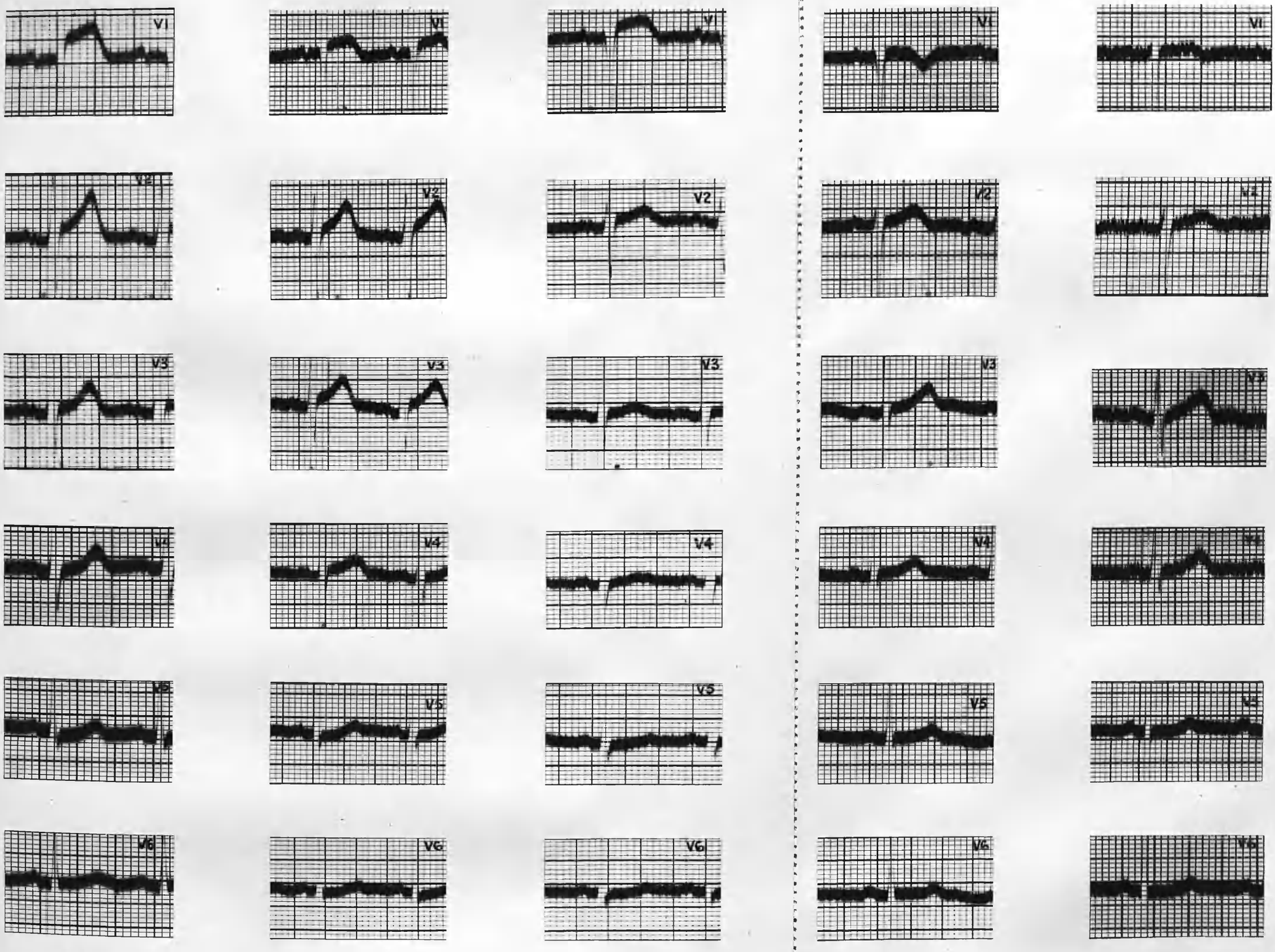
2 DAYS
AFTER ONSET.

5 DAYS
AFTER ONSET.

12 DAYS
AFTER ONSET.

4 WEEKS
AFTER ONSET.

5 WEEKS
AFTER ONSET.



CASE 49, H.R., MALE 60 YEARS.

2 DAYS AFTER ONSET.

5 DAYS AFTER ONSET.

12 DAYS AFTER ONSET.

4 WEEKS AFTER ONSET.

5 WEEKS AFTER ONSET.

Case 55. H.J., 57 years, male.

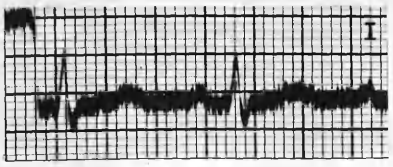
Patient was admitted to hospital on the day following the onset of praecordial pain which struck him while walking along the street to work. At first the pain radiated down both arms to wrists and also to the interscapular region and the back of the neck. With some difficulty he returned home and thereafter collapsed. The total duration of pain was about twelve hours in the first instance but for several days after admission there were slight recurrences of cardiac pain. There was slight pyrexia for several days. Blood pressure which on admission was 132/90 fell to 110/74, then to 108/72 two days later at which level it remained for several weeks. On the fourth day of his hospital stay, pericardial friction was heard over the praecordium, it continued for five days. The cardiac sounds were otherwise of poor quality. Radiological examination of the heart showed slight enlargement chiefly affecting the left ventricle.

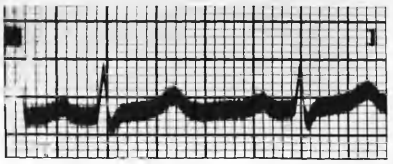
A twelve-lead electrocardiogram obtained eleven days after onset shows the typical signs of a recent posterior infarction in the standard limb leads. Lead VF shows a deep QS deflection which might have been a normal

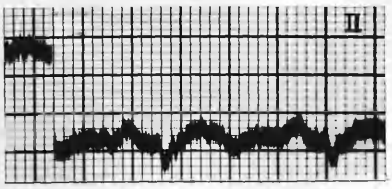
variant in a horizontal heart but closer inspection shows a broad late R wave just rising above the isoelectric level; combined with the ST elevation and T inversion there is no doubt that infarction has occurred in the posterior wall. Lead VL is the reciprocal of lead VF. Lead VR shows conspicuous downward bowing of the ST segment which is typical of this lead when it faces an infarction in the anterolateral or posterolateral wall. The chief features of the praecordial leads are the splintering and breadening of the RS deflection in leads V1 and V2 and the inconspicuous T waves throughout. The former may be due to uneven or patchy contraction in the cardiac wall opposite the site of the electrode and the homologous with the small splintered QRS of standard limb lead I; the latter may be due to the pericarditis of the anterior cardiac wall which was present clinically and is in striking contrast to the exaggeration of the T waves which is usual in the anterior praecordial leads in posterior infarction. Similarly the height of the R wave does not suggest reciprocal exaggeration.

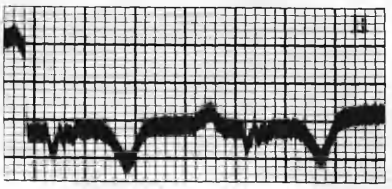
The second electrocardiogram taken a week later, that is eighteen days from onset, shows the usual evolution of ST-T signs in the standard and unipolar limb leads. The

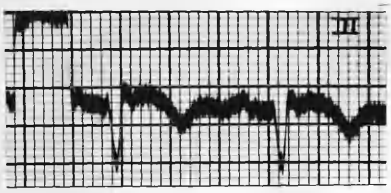
chief change in the praecordial leads is the increased height of the T waves due partly to the resolution of the anterior pericarditis and partly to the usual evolution of the T waves in posterior infarction. There is no significant change in the QRS complexes. This case is an example of a posterior infarction complicated by anterior pericarditis.

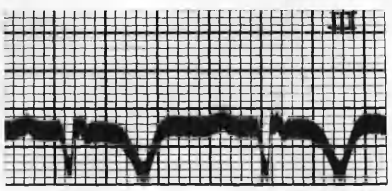


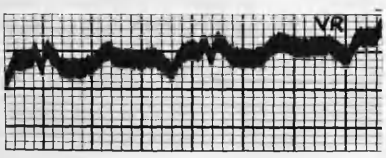


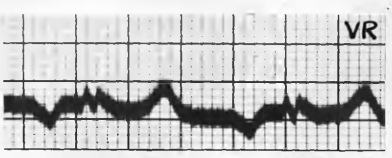


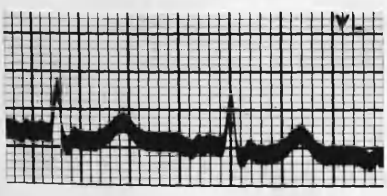


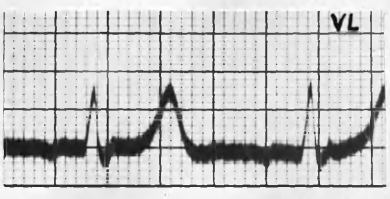


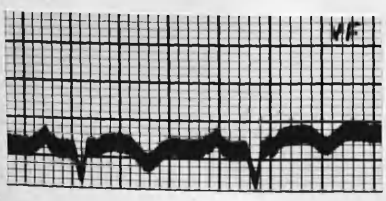


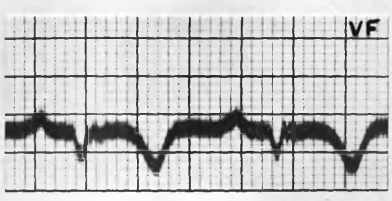








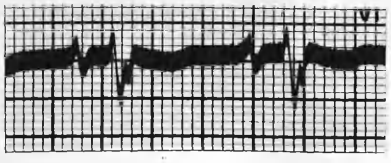


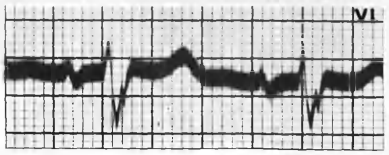


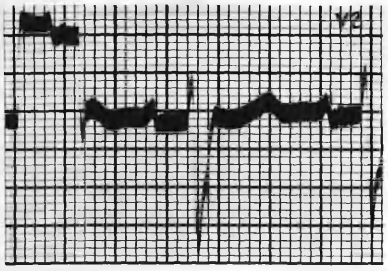
CASE 55 H.J., MALE 57 YEARS.

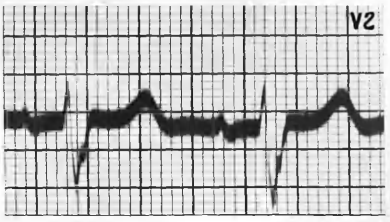
11 DAYS
AFTER ONSET.

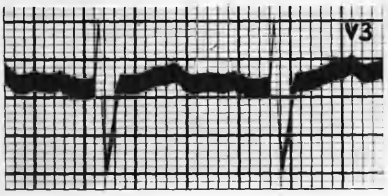
18 DAYS
AFTER ONSET.

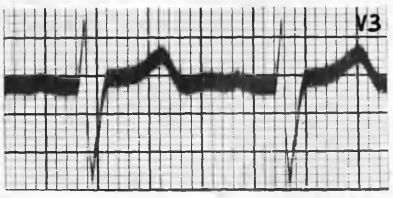


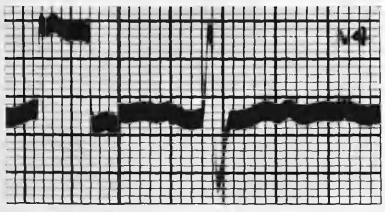


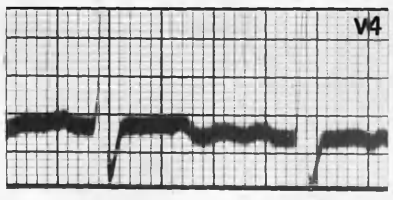


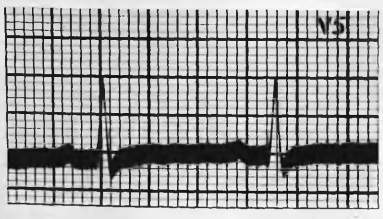


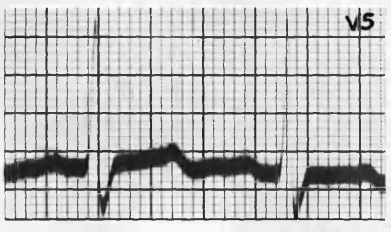


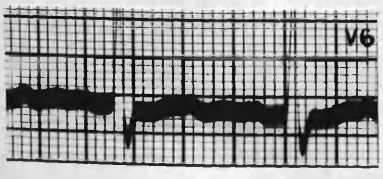


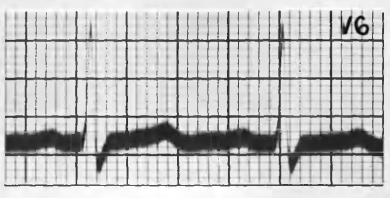












CASE 55 H.J., MALE 57 YEARS.

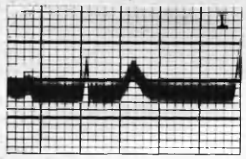
11 DAYS
AFTER ONSET.

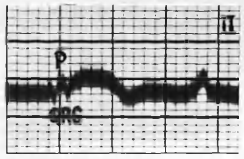
18 DAYS
AFTER ONSET.

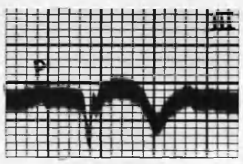
Case 56. J.B. Male, 48 years.

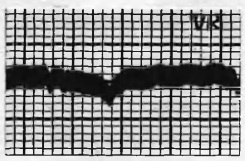
This patient collapsed with gripping substernal pain while at work. Blood pressure on that day was 80/50; the heart sounds were poor and a leucocytosis occurred. The electrocardiogram herewith was obtained one week after onset. There is complete heart-block. The classical signs of recent posterior infarction are present in the standard limb leads. The QRS deflection of lead VF however is a small splintered deflection largely negative in direction but barely reaching 0.5 millivolt which is the minimum diagnostic requirement. However there is a frank elevation of the ST segment. Nevertheless lead VF does not add any further diagnostic sign in this case; in actual fact none is required. Lead VL shows absence of the S wave, slight depression of the ST segment and a sharply upright T wave, all of which are commonly found in lead VL when it apposes the endocardial surface of a large infarct. Praecordial leads V2 to V6 show slight depression of the ST segment which is typical of these leads when in apposition to a large posterior infarction. Towering /

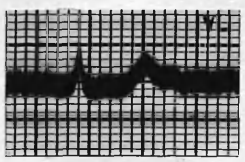
Towering spike-like T waves have already developed in leads V2, V3 and V4. The QRS formation of the anterior praecordial leads, Viz. unduly tall R waves and shortened S waves is due to the reciprocal effect of the infarct in a horizontal heart with counter-clockwise rotation so that the electrocardiographic pattern before infarction already showed a tendency to a left ventricular type.

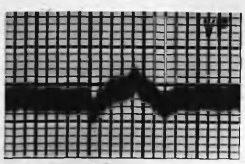








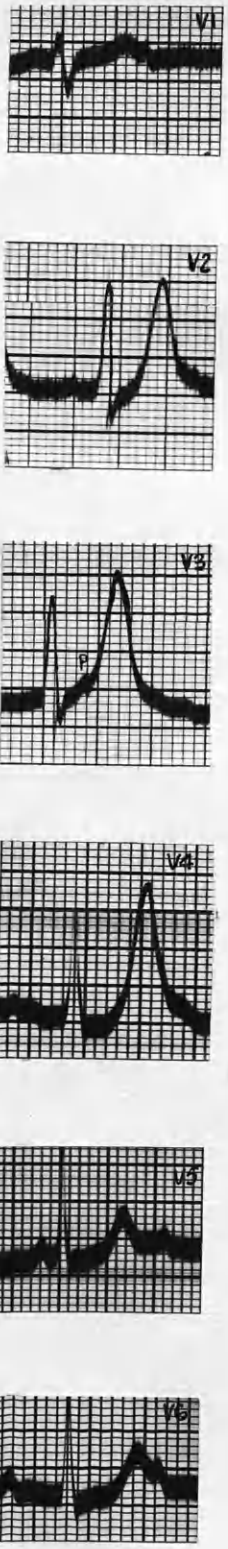




CASE 56, J.B. MALE, 48 YEARS.

1 WEEK

AFTER ONSET.



CASE 56, J.B. MALE, 48 YEARS.

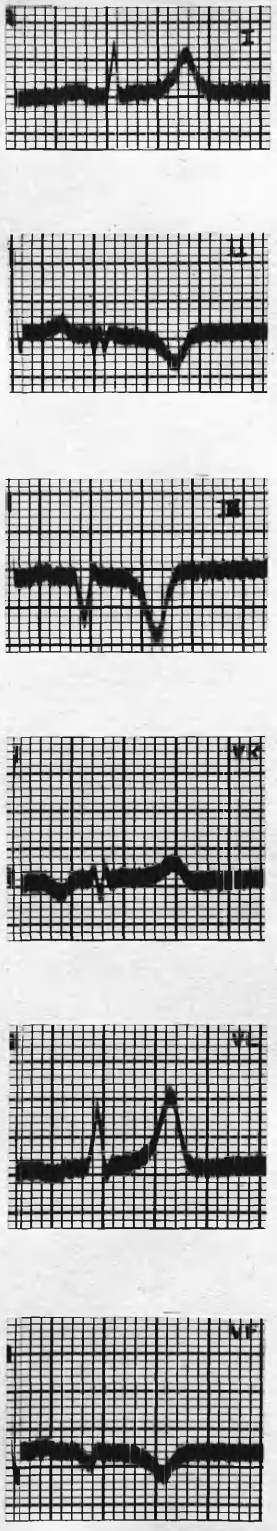
1 WEEK

AFTER ONSET.

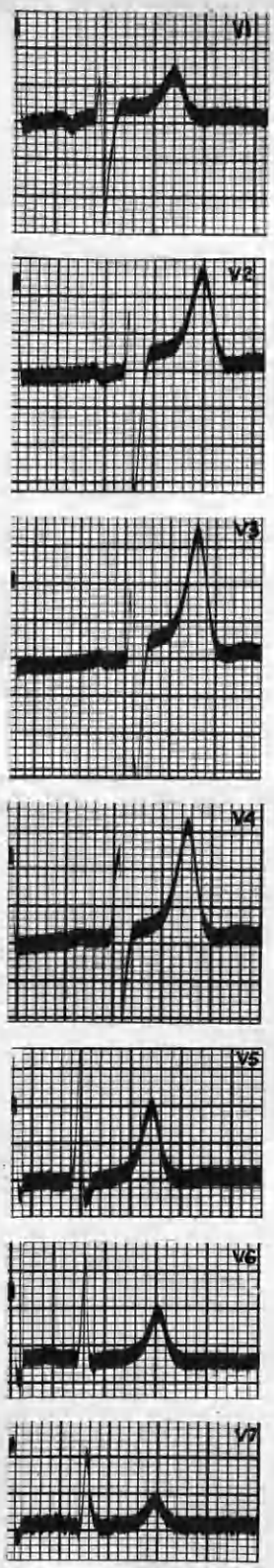
Case 57. W.N. Male, 49 years.

This patient was examined four weeks after the onset of chest pain which had lasted seven hours. Records by his own practitioner showed blood pressure of 120/70 or thereby throughout. There was no cardiac enlargement but the cardiac sounds were soft. An electrocardiogram shows the typical signs of an organising posterior infarction in the standard limb leads. Lead VF shows a small splintered QRS deflection which is almost entirely negative in sign and which fails to reach the minimum 0.5 millivolts required for diagnosis. The ST segment is a plateau and the T wave shows cove-plane inversion. In spite of its small size lead VF clearly records potentials from the epicardial surface of an infarction. Lead VL is typical of the left arm lead when it apposes the endocardial surface of a severe posterior infarction. Deprived of potentials normally generated by this portion of the posterior wall there is apparent exaggeration of positive deflections in this lead - hence the prominent and slurred R wave and the very large symmetrical T wave. The sharply upright /

/ upright T wave of lead VR is also attributable to infarction. The praecordial leads are within normal limits for their RS deflections but their T waves are unduly prominent especially in leads V2 to V4 where they are tall and spike-like. The praecordial leads would appear to be in apposition to the zone of ischaemia around the infarction, but not to the infarct itself.



CASE 57, WN. MALE, 49 YEARS.
4 WEEKS AFTER ONSET.



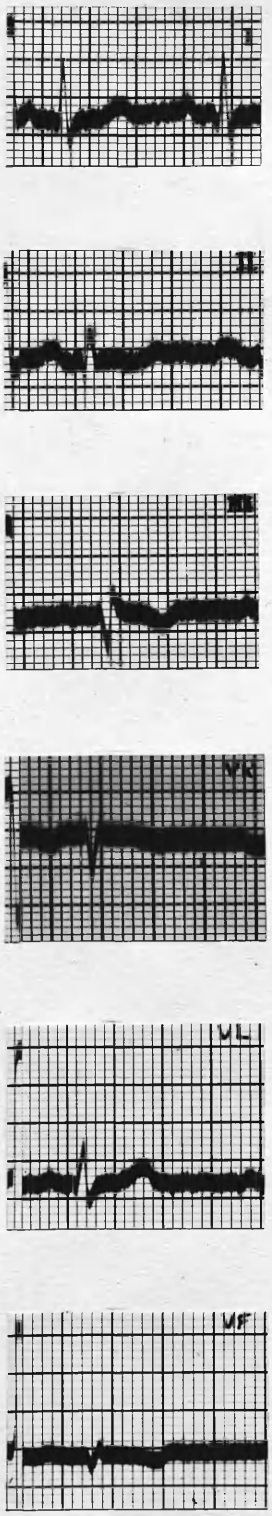
CASE 57, W.N. MALE, 49 YEARS.
4 WEEKS AFTER ONSET.

Case 58. G.F. Male, 44 years.

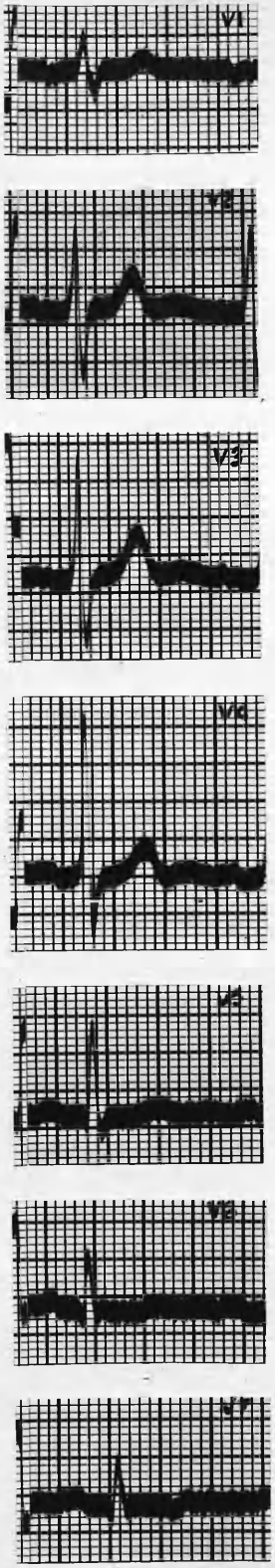
This patient attended the out-patient department ten weeks after a clinical attack of myocardial infarction for which he had been treated at home. The original cardiac pain had recurred over a period of two weeks. When examined as an out-patient, he presented no abnormal physical sign in heart or lungs. An electrocardiogram shows persistent signs of posterior infarction in the standard limb leads. Q waves are present in leads 2 and 3; the ST segments are convex upwards and the T waves are inverted. Lead VF shows a QR deflection with a Q/R ratio of 1 but the entire deflection is small and barely reaches the size of 0.5 millivolt which is the minimum diagnostic requirement. The ST segment is slightly shouldered and the T wave is diphasic. In spite of its small size, lead VF is clearly confirmatory of infarction, if confirmation is necessary. Lead VL displays a prominent R wave and small S wave with sharply upright T wave. While this pattern may indicate no more than a horizontal, semihorizontal or intermediate position, the shortened S wave may /

/ may be partly attributable to the reciprocal effect of ~~the~~ posterior infarction. The praecordial leads show that V7 overlies the edge of the infarct - there is a significant Q wave and upward convexity of the ST segment. The infarct is therefore posterolateral rather than posterior. Praecordial leads V1 to V5 are strictly within normal limits for an intermediate to horizontal heart but in view of diagnosis of posterior infarction, already made, it is probable that the R waves of leads V1 to V3 are unduly tall and their S waves shortened as a reciprocal effect of the infarction in the posterior wall.

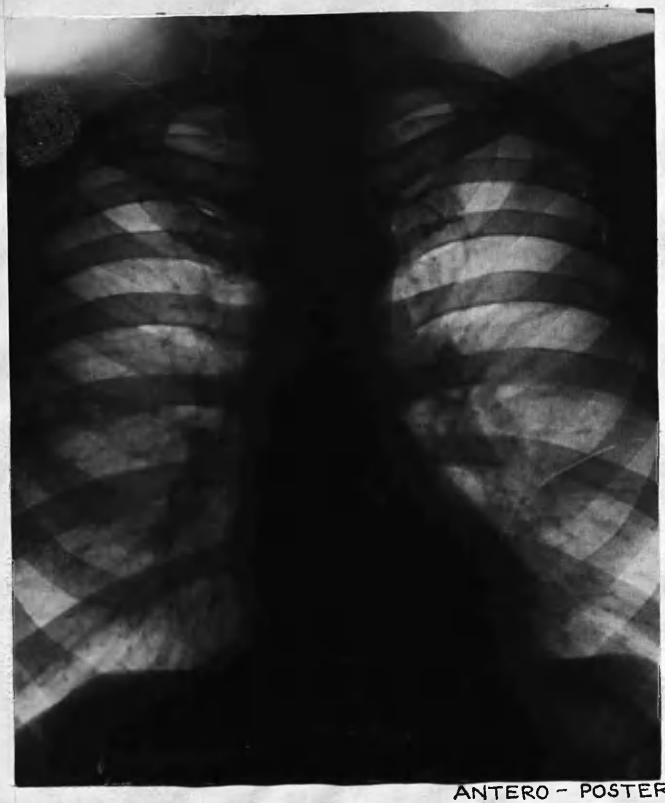
This case shows that while the unipolar leads may be superfluous in diagnosis, they give some indication of the size of the infarction and explain the patterns of the standard limb leads in terms of cardiac position and situation of infarction.



CASE 58 G.F MALE, 44 YEARS.
10½ WEEKS AFTER ONSET.



CASE 58 G.F. MALE, 44 YEARS.
10½ WEEKS AFTER ONSET.



ANTERO - POSTERIOR

LEFT ANTERIOR OBLIQUE

Case 59. F.F., male, 45 years.

This patient had an attack of constricting substernal pain lasting for several hours about two weeks before he was referred to the Out-Patient department. Blood pressure was 140/80. There was no clinical enlargement of the heart and the cardiac sounds were of average quality.

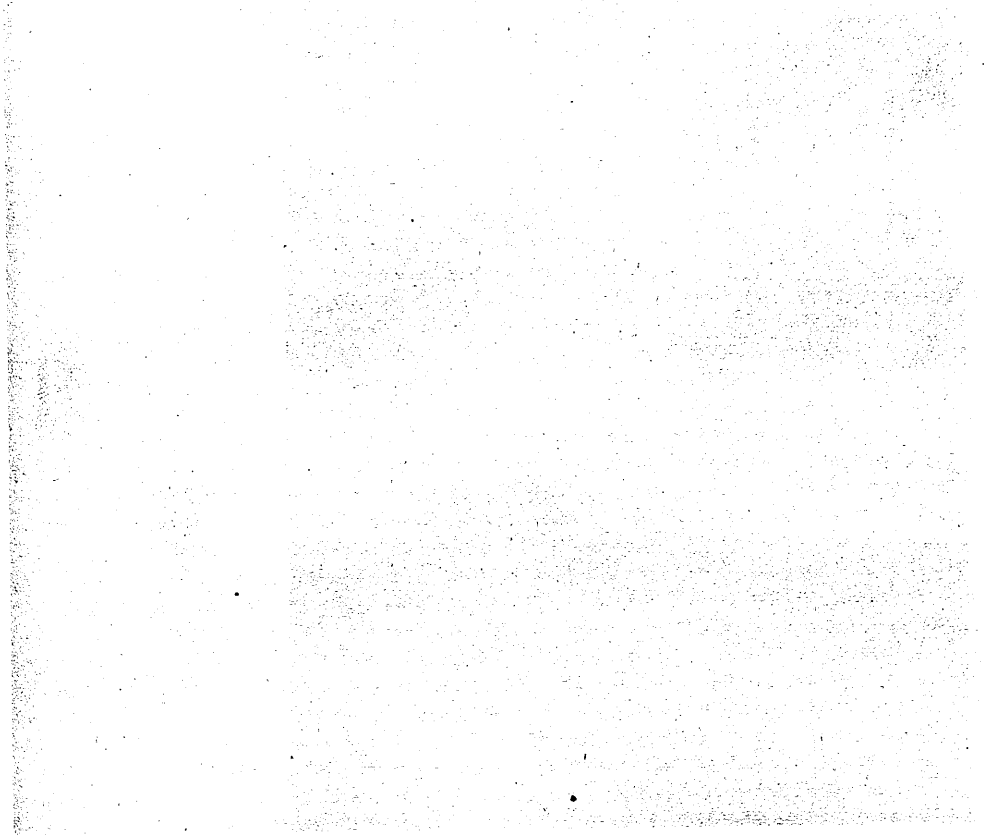
An electrocardiogram shows equivocal standard limb leads. There is no Q wave in lead 1 but there is upward convexity of the RS-T segment and shallow inversion of the T wave, such as may be found in anterolateral or posterolateral infarction. Standard limb lead 2 shows slight upward bowing of its RS-T segment. The third lead also presents dubious signs, viz. a splintered QS complex and slight elevation of the RS-T segment. While these leads undoubtedly raise the possibility of infarction they do not present a typical pattern nor do they eliminate the diagnosis of pericarditis. The unipolar limb leads show a small splintered QS in the left thigh lead, which can be adequately explained either by incomplete transmural infarction of the posterior wall of the left ventricle if the heart is in the intermediate position or by infarction of the septum in a horizontally placed heart. The latter alternative is favoured since infarction of the posterior wall of a fortnight's age is more usually represented by a QR than by a short splintered QS deflection, the R wave being derived from intact regions of the posterior wall. However the decision that the splintered QS of /

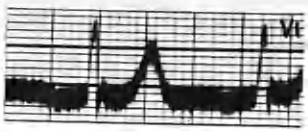
/ of lead 3 is indicative of septal infarction does not eliminate involvement of the neighbouring part of the posterior wall in addition. Furthermore the axillary leads show upward convexity of their ST segments and inversion of their T waves which would suggest ischaemia of the lateral ventricular wall or patchy intramural infarction, probably an extension from the posterior part of the infarction. The tiny Q wave and sudden fall in the height of the R wave in lead V6 is in favour of patchy lateral infarction rather than simple ischaemia.

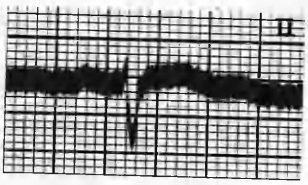
Further indirect evidence that the heart is in the horizontal position is gained by a consideration of the anterior praecordial leads. Their tall R waves, shortened S waves and peaked symmetrical T waves indicate the reciprocal effects of a posterior infarction, superimposed on a left, not right, ventricular pattern, such as is recorded by these leads in unusual degrees of counterclockwise rotation which may occur in horizontal hearts. Rotation about a vertical axis is also demonstrable by the QR form of lead VR which is derived from the posterobasal region of the left ventricle due to backward rotation of the apex. Lead VL is more difficult to interpret with its RS, R^1 complex, slightly shouldered ST and shallow inverted T wave. In view of the counterclockwise rotation it is probable that this pattern represents potentials from the upper marginal zone of the posterolateral infarction.

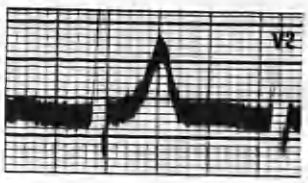
Thus the unusual cardiac position is responsible for the unexpected patterns recorded by the unipolar limb /

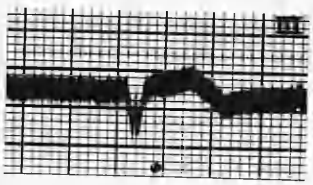
/ limb leads and therefore by the standard limb leads. The praecordial leads conform to a more usual configuration and there are on the whole reasonable grounds for the localisation of the infarction to the posterolateral and posteroseptal walls.

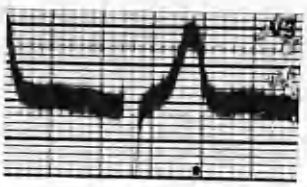


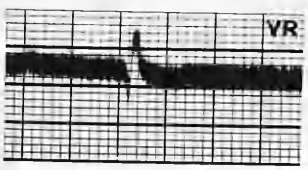


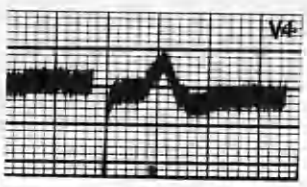


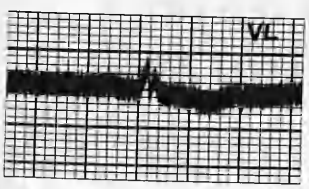


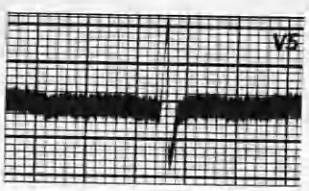


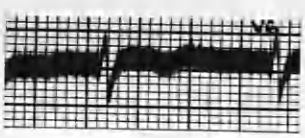












CASE 59 F.F., MALE 45 YEARS.

2 WEEKS
AFTER ONSET.

A.

COMPARISON of STANDARD LIMB LEADS and UNIPOLAR LEADS in the DIAGNOSIS of POSTERIOR INFARCTION

The advent of unipolar electrocardiography has advanced the accuracy of diagnosis of posterior infarction in so far as the analysis of standard limb lead III, provided by the left arm and left thigh unipolar leads, has made it possible to separate off those cases which show a coronary type of lead III as a normal variant from those in which it signifies infarction. Criteria for the diagnosis of infarction are now applied to the left thigh lead and no longer to standard limb lead III. According to Goldberger (1945) a Q wave in lead VF which has a duration of 0.04 sec. (from onset of the QRS deflection to where the upstroke of R crosses the base-line) and an amplitude 60% of that of the succeeding R wave or 40% of that of the entire QRS complex is diagnostic of posterior infarction. The standards described by Myers et al (1949) are more comprehensive, viz. the QR complex of lead VF must measure 0.5 millivolt or more, the Q wave must have a duration from onset to nadir of 0.03 sec. and the Q/R ratio must be over 25%. Furthermore, Myers et al (1949), have established that while oesophageal

/ oesophageal leads may be presumed to be in closer relation to any posterior infarction than any external lead, and therefore to be superior in diagnosis, yet there is close conformity with the findings in the left leg lead.

It must be clearly recognised that difficulty in diagnosis arises only in a minority of cases. Of the twenty-eight cases of posterior infarction in the present series, twenty-two show signs more or less diagnostic of infarction in standard limb leads II and III, and, in them, lead VF is purely confirmatory and, not infrequently, redundant. This high proportion emphasises the fact that the pattern of infarction recorded by the left thigh lead is but little mitigated by the reciprocals of the right arm and left arm leads. Far from lessening the diagnostic features of lead VF, the left arm lead in reciprocal may actually augment them, especially if it faces the endocardial surface of a large infarction.

The group of six cases in which the standard limb leads failed to establish the diagnosis of posterior infarction provides an opportunity to evaluate lead VF and the other unipolar leads in diagnosis. Three of the six cases (Nos. 43, 44, 45) show specially tall R waves in standard limb leads II and III and also in lead VF so that the Q/R ratio in lead VF falls below one quarter which is the minimum required in the diagnosis of infarction. Furthermore the ST-T changes are equivocal so that the /

/ the diagnosis cannot be based on lead VF. All three cases have vertical or semivertical hearts; hence, facing, as it does, the left ventricle, lead VF tends to show a tall R wave and therefore any Q wave which is the result of infarction may not be large enough to make the Q/R ratio significant. The position is further complicated by the fact that in vertical and semivertical hearts a septal Q wave may be registered by lead VF. Furthermore the infarct may be small in surface area and thus there is little reduction in the height of the R wave in lead VF or the infarct may be in a part of the posterior wall less advantageously placed to affect the potentials of the left leg. Infarcts of the middle third exert the greatest influence on lead VF; those of the apical third and of the basal third in that order in lessening degree. (Myers et al 1949). Thus physiological and topographical considerations may outweigh those due to infarction.

Thus from the point of view of early diagnosis in these cases lead VF does not supply the key. Nor is much help to be derived from leads VR and VL. Lead VR in vertical and semivertical hearts is somewhat remote from the electrical disturbance caused by small posterior infarcts and does not as a rule reflect changes derived directly from its endocardial or pericardial surfaces. Lead VL although in closer relation to the infarct than VR is unlikely to be severely affected reciprocally by infarcts whose effects are /

/ are relatively limited even in lead VF. Nevertheless in the three cases cited lead VL shows shortening of its S waves but this fact can only be appreciated in retrospect. It is in these cases that the sequential changes of the ST-T segment are of high diagnostic value, not only those shown by lead VF and therefore by standard limb leads II and III, but also those shown by leads VR and VL and the praecordial leads. In lead VF and in leads II and III, the ST segment may become flat from a previously dubious bowing and cove-plane T waves may develop. Tall spike-like T waves may be present originally or may emerge in the anterior praecordial leads and are typical of an organising posterior infarction (cases 44, 45).

The fourth case (No. 46) is very similar to the preceding three cases - a small subendocardial infarction has occurred in a semivertical heart. Although standard limb lead III is of coronary type, (Q/R ratio is 1, the ST segment is a plateau and the T wave is sharply inverted), it is not supported by adequate signs in lead II and the diagnosis cannot be based on these leads. Lead VF however just succeeds in fulfilling the criteria of Myers, as discussed in the text of the report. The tall R wave which is typical of the semivertical position is not so tall as that of the preceding cases so that the Q/R ratio is higher, viz. 1/4, and there is slight delay in attaining the peak of the R wave. The ST-T segment and T wave give little /

/ little supporting evidence. However, while arithmetical limits for the Q/R ratio are unavoidable it would be futile to maintain that the diagnosis of infarction can be based with certainty on lead VF in this case although not in the preceding three. It is probable that all belong to the same category, viz. vertical or semivertical hearts with small subendocardial infarctions. As in the foregoing cases there is confirmatory evidence of posterior infarction in the spike-like T waves in the anterior praecordial leads and in the subsequent sequential changes of healing.

The fifth case (No. 34) is in a different category and is one of the two cases where, in the presence of equivocal standard limb leads, the unipolar limb leads did supply the diagnosis of posterior infarction. In case No. 34 standard limb lead III is of coronary type but there are insufficient signs in lead II and the possibility cannot be eliminated that lead III is a normal variant in a horizontal, semi--horizontal or intermediate heart. The unipolar limb leads clarify the position however. In spite of the fact that the QRS deflection of lead VF is less than 5 mm. high and is therefore of limited diagnostic value, the diagnosis of infarction is at least strongly suggested by the plateau form of the ST segment, and, in lead VL, by the depression of the ST segment, which is typical of a lead opposing the endocardial surface of an infarct. While the validity of the diagnosis of infarction may be criticised on the grounds /

/ grounds of the small size of the ventricular deflection of lead VF and the absence of a Q wave, remaining doubt is removed by the subsequent changes seen in lead VF. By two and a half days after onset, its QRS deflection reaches 0.5 millivolt and two days later, a Q wave has appeared; presumably the increase in size of the R wave is due to some recovery of potentials at first severely ablated by the severity of the infarction.

The sixth case (No. 59) represents a different problem, viz. that, while the ST-T abnormalities of all three standard limb leads raised the possibility of infarction, yet the QRS complexes are equivocal. Lead III alone presents a Q wave. As described in the text the unipolar limb and praecordial leads localise the infarction to the posteroseptal and posterolateral regions of the left ventricle. The ambiguity of the standard limb leads is due to an unusual cardiac position, viz. horizontal with a severe degree of counterclockwise rotation and backward rotation of the apex.

These cases illustrate the limited value of lead VF in diagnosis in vertical or semivertical hearts where the infarction is either subendocardial or is in a part of the posterior wall which has little influence on the potentials of the left leg but the unipolar limb and praecordial leads are nevertheless essential in establishing cardiac position in the first instance before evaluating the signs, limited

/ limited though they may be, in lead VF and in the other unipolar leads. They also emphasise the value of sequential changes in the unipolar leads in the diagnosis of infarction and that atypical signs in the standard limb leads may be explained by an assessment, not of one, but of all nine unipolar leads.

SUMMARY

In twenty-two out of twenty-eight cases of posterior infarction, the standard limb leads are sufficient for the diagnosis. In five of the remaining six cases, a single recording of lead VF is of little diagnostic value and in the sixth such a tracing just succeeds in fulfilling accepted diagnostic criteria of infarction. In the five cases, a single record of lead VF shows a QRS deflection which is within normal limits in three and is, theoretically, too small to be of diagnostic value in two. Nevertheless the changes exhibited by the other unipolar limb and praecordial leads, derived directly or reciprocally from the infarction and the sequential changes shown by all the unipolar leads (VF included) leave no doubt that posterior infarction is present.

UNIPOLAR ELECTROCARDIOGRAPHY as an AID to DETERMINING SITE, EXTENT and HEALING of POSTERIOR INFARCTION

The foregoing section has demonstrated some limitations in the diagnostic value of lead VF: in no case out of the series of twenty-eight does the diagnosis of posterior infarction rest on lead VF alone, and among the six where the standard limb leads are equivocal, only one shows signs in lead VF just acceptable as indicating infarction. The possibility must be explored however that lead VF may supply information regarding the exact site of the infarct and the process of healing. Myers (1949) has established that signs diagnostic of infarction are most likely to occur in lead VF if the infarct is in the middle third of the posterior wall; they are less likely if the infarct is in the apical third and least likely if it is in the basal third. However the present study, being largely electrocardiographic, adds nothing to these particular topographical subdivisions.

Six (Nos. 28, 32, 35, 41, 49, 55) of the twenty-eight cases have QS deflections in lead VF. This is a normal finding in horizontal or semihorizontal hearts but the presence of the infarction disturbs the signs by which electrical cardiac position is determined and only a broad

/ broad assessment is possible. In one of the six cases (No. 28) this difficulty has been obviated by the later radiological finding of the paradoxical pulsation of an aneurysm of the posterodiaphragmatic wall, so that the QS deflection of lead VF in this case may unequivocably be attributed to the large transmural infarction of the posterior wall. In the other five cases, (as well as in No. 28) a conspicuous R wave in lead VL proclaims an intermediate to horizontal position of the heart and in two (Nos. 32 and 41) the praecordial leads would suggest left ventricular hypertrophy or dilatation, and hence in these two instances at any rate, the horizontal or semihorizontal position cannot be eliminated as the explanation of the original QS deflection of lead VF. Even when it is accompanied by the RS-T signs of infarction, the ascription of the QS to infarction is based on presumption not deduction; if it is due to infarction then the site of the infarction is the posterior part of the septum (Myers, 1949).

If however the QS deflection of lead VF becomes a QR deflection in later tracings and if this development is not attributable to changes in electrical position, then the original QS may rightly be attributed to infarction, the advent of the R wave signifying some restoration of function, whether the infarction is situated in the posterior part of the septum in horizontal or semi--horizontal hearts or in the free posterior wall in /

/ in intermediate hearts. All six cases develop a small late R wave after intervals varying from not less than a few weeks to several months after infarction. Myers points out that the QS deflection of lead VF which is due to infarction of the free posterior wall is a transient phenomenon, for very soon an R wave is provided by unaffected portions of the posterior wall. However the shortest interval between onset and a QR deflection in the present six cases is seen in No. 35 where it is three and a half weeks so that the delayed appearance of the R wave lends favour to the view that the infarction involved the septum and was not confined to the free posterior wall.

In eleven (Nos. 27, 29, 31, 33, 36, 38, 39, 42, 47, 48, 58) of the twenty-eight cases, a typical QR deflection is recorded by lead VF from the beginning; all measure five millimetres or more, the Q/R ratio is at least 1/4 and all have significant RS-T sequential changes; infarction of the free posterior wall is justified whether the R wave is derived from intact subepicardial muscle or from unaffected portions of the posterior wall. In one case (No. 39) the QR deflection gives place to a deep splintered QS deflection, signifying spread of infarction as described in the text of the report; subsequently a QRS deflection is again established comparable to the foregoing group. In another case (No. 42) a QR originally five millimetres in height becomes reduced to three millimetres and, although outwith /

/ outwith diagnostic usefulness as a result, is consistent with the deterioration in electrocardiographic signs in other leads, as explained in the text.

In general, the QR complex of lead VF whether it was present originally as such, or whether it developed out of a QS deflection, shows little change in later tracings and in them the picture of posterior infarction becomes fixed in the standard limb leads also. When a posterior infarction heals more satisfactorily, lead VF may provide evidence of its previous presence, although the residual signs in the standard limb leads are equivocal. It is documented as a significant Q wave, delay or slurring in attaining the peak of the R wave and usually a flat ST-T segment, signs which indicate a conduction defect in the posterior wall of the left ventricle. Case 33 exemplifies these features in the tracing taken one year and ten months after onset. Standard limb leads I and II are normal; the small Q wave of lead II may gain significance when viewed in conjunction with the considerable Q wave of lead III but the latter is largely the reciprocal of the positive R wave of lead VL, the heart being semihorizontal or intermediate. Thus lead VF alone provides signs which indicate a chronic focal lesion in the posterior wall. Similar conduction defects in lead VF are seen in three other cases (Nos. 29, 39, 42) although in these instances the standard limb leads are more or less suggestive of previous posterior infarction.

Of /

/ Of the original series of twenty-eight, seven cases (Nos. 30, 34, 37, 40, 56, 57, 59) have in their first records QRS deflections in lead VF which are less than five millimetres in height (one millivolt = ten millimetres) and other four cases (Nos. 27, 33, 35, 42) develop such small deflections during the second, third or fourth weeks after onset. Thus they do not conform to the minimum requirement of the criteria of both Goldberger (1945) or Myers (1949). While such small deflections are prone to fallacious interpretation, the associated elevation of the ST segment and T changes are nevertheless sufficiently definite to make such a QRS of some diagnostic value, for its very smallness may be significant of failure of the left ventricle to develop adequate potentials, so much of its wall being involved in the infarction. The persistence of such a small QRS deflection indicates poor healing although a minute R wave may re-appear, to indicate minimal local functional recovery in the muscle subtended by the electrode on the left leg. The final picture is that of a conduction defect in the posterior wall, probably severe in cases 30, 37, and 40 and of lesser degree in case 34. These cases illustrate the value of lead VF in detecting and estimating residual lesions following posterior infarction.

In spite of very small QRS deflections in lead VF, three of the above seven cases (Nos. 37, 56, 57) have the classical signs of posterior infarction in the standard /

/ standard limb leads. This is partly due to the fact that, although the QRS is small, the ST-T changes are prominent and are, in fact, reinforced by the reciprocal of the RS-T changes of lead VL in the formation of standard limb lead III and in one of the three cases (No. 57) by a similar summation of effects with lead VR in standard limb lead II. But it is clear however that standard limb leads II and III have depended more on the reciprocals of leads VR and VL respectively for their QRS patterns than on the direct transference of the small QRS of lead VF. Hence it is important to assess to what extent these leads have been affected by the presence of the posterior infarction, not only in those cases where the QRS of lead VF is small but also in those with larger VF deflections, although in the latter the influence of lead VF in the formation of leads II and III is all the greater.

Lead VR is somewhat remote from the electrical disturbances emanating from a posterior infarction and as a rule there is no recognisable alteration of the rS, QS, rSr' or QR complex normally recorded by lead VR. Seven cases of the present series (Nos. 27, 33, 35, 36, 38, 42, 47) have rS deflections, the r being derived from activation either of the left side of the septum or of the anterior cardiac wall. This pattern is common in vertical, semivertical and intermediate hearts especially, with moderate clockwise rotation around the heart's long axis and forward rotation /

/ rotation of the apex around a transverse axis. In reciprocal, the small r wave of lead VR exaggerates the Q wave of lead VF in the formation of standard limb lead II. Furthermore the R wave of lead II is largely provided by the S wave of lead VR for, in all seven cases cited, the R wave of lead VF is very small. It is probable that in these cardiac positions the small size of the R wave of lead VF is just as much the result of the infarct as the presence of the Q wave; hence the relative smallness of the R wave of lead II is as much a sign of infarction as the large Q wave of that lead.

In two cases out of the series of twenty-eight the QRS complex of lead VR is affected specifically by the presence of the infarction and thus further information regarding its extent is provided. In case 40, while the patient was under observation, the pattern of infarct developed in lead VR - a significant QR deflection (not attributable to change in cardiac position), shouldering of the ST segment and inversion of the T wave. As explained in the text, the probable interpretation is extension of the infarction into the interventricular septum, the reference of septal potentials to the right arm being no doubt aided by the vertical position of the heart. Septal extension is also the explanation offered for the abnormal pattern of lead VR in case 34 where there is no r wave, only an Sr', until healing is well advanced. In the early /

/ early tracings there is no Q wave in lead VF so that there is no Q in standard limb lead II but even when a small Q develops in lead VF it is engulfed by the immediate negativity of the right arm lead in reciprocal. When the septal extension has healed the r wave of lead VR returns and likewise the Q of lead II.

The RS-T deflections of lead VR are much more frequently influenced by the presence of the infarction, in particular of its endocardial surface. In such cases the electrode of the right arm is in apposition to extensions of the posterior infarction into the septum or into the adjacent wall of the right ventricle, irrespective of cardiac position. The signs in lead VR are closely similar to those exhibited by this lead when it is in apposition to a large anterior infarction; they are exemplified by eight cases (Nos. 27, 31, 35, 41, 42, 47, 55, 57). In early tracings there is depression or down--ward convexity of the RS-T segment and, by the end of the second week after onset, the T wave is large, symmetrical and upright, similar to the T waves of the anterior praecordial leads. In well healed infarctions the T wave of lead VR may return to a normal inverted form.

The changes in lead VL are more constant and more specific than those in lead VR because the former lead is frequently in direct relationship to the endocardial surface of the infarcted region of the posterior wall. In

/ In view of the fact that there is a much closer spatial relationship between this region and lead VL than between the epicardial surface of the infarct and lead VF there is a strong prima facie case for obtaining signs of comparable diagnostic importance from lead VL as from lead VF. A typical pattern is seen in lead VL in one third of the cases (nine out of twenty-eight cases, Nos. 27, 28, 30, 35, 36, 41, 47, 55, 57), all of which show an intermediate to horizontal position. The R wave is conspicuous; there is no preceding Q wave and the rise of the R wave to its peak, denoting the onset of the intrinsic deflection, is immediate and steep. It would appear that the muscle subtended by the electrode on the left arm, presumably intact muscle, is activated very early in ventricular contraction. There is no evidence that the prominence of the R wave is due to artificial exaggeration caused by failure of potentials normally antagonistic to it, for in no case does it diminish in size with healing of the infarction. This sudden R wave accounts in large measure for the deep Q wave of standard limb lead III for, in all nine cases, it is larger than the more delayed Q wave of lead VF. The downstroke of the R wave may be thickened or slightly notched or it may be followed by an abortive or truncated S wave, rarely reaching below the isoelectric level. These oscillations are clearly a reflection of the delayed potentials arising in more remote parts of the ventricle, probably in the region of the infarct. In reverse /

/ In reverse, they usually coincide with the small delayed R wave of lead VF but, even when summated in standard limb lead III, these R waves are still of very small size.

Whether or not the above QRS pattern is displayed, the RS-T formation invariably shows the influence of the endo--cardial aspect of the infarcted area - the RS-T segment shows downward convexity and subsequently there is a prominent symmetrical upright T wave. These features are commonly found in any of the three unipolar limb leads when the electrode is apposed to an infarct in the remote ventricular wall.

The absence of a Q wave in lead VL, noted above, is a feature of all twenty-eight cases of the present series with one exception (No. 38). The absence of Q must not be construed as an indication of septal involvement, for the retention of a prominent septal Q wave in the axillary leads is seen in several cases (Nos. 28, 32, 35, 36, 41, 47, 58). Rather should it be interpreted along physiological lines, viz. that the spatial relationship of the septum to the left arm is such that septal potentials are active in a plane at right angles to the electrode on the left arm which, therefore, fails to record them. The absence of a Q wave from lead VL decides that there is none in standard limb lead I unless a small normal R wave (in reciprocal a Q wave) is furnished by lead VR. While such an R wave is seen in several cases (Nos. 27, 28, 31, 33, 35, 41, 42, 47, 57) in

/ in none of them is it transmitted as a Q wave to standard limb lead I because the positivity of the left arm is so immediate and increasing that the transient negativity derived from the right arm merely causes a slight thickening of the upstroke of R in standard limb lead I. This immediate negativity of lead VL and its effects constitutes, in the opinion of the present writer, a very important sign of uncomplicated posterior infarction. The exception mentioned, viz. case 38, emphasises the premise; lead VL shows at fifteen months a QR pattern of a type suggesting a local conduction defect so that the ventricular wall subtending the electrode, probably the upper anterolateral wall, is actually involved in the ultimate fibrotic process. When this Q wave is added to the R wave of lead VR there is a considerable Q wave in standard limb lead I. In this case the lesion has been by no means confined to the posterior or posterolateral wall. Two other cases (Nos. 36, 39) show small Q waves in standard limb lead I, both derived from lead VR and both emerging in lead I because the positivity of the left arm is delayed, in the one case (No. 39 twenty months after onset) by a severe conduction defect in the lateral wall and in the other (No. 36) because the electrode on the left arm is in direct apposition to muscle close to the edge of the infarction - the heart is horizontal with severe counterclockwise rotation and the infarct is posterolateral. Signs /

/ Signs of healing are well seen in leads VR and VL especially in vertical or semivertical hearts. In these cardiac positions leads VR and VL tend to record the negative potentials of the ventricular cavities documented by S waves in these leads. During the acute stage of infarction these S waves are small but they elongate with satisfactory healing of the infarction. (cases 34, 40, 43, 44, 45, 46). This process is parallel to the elongation of the S waves seen in the anterior praecordial leads. When the heart is horizontal or semihorizontal however, the electrode on the right arm may not face into the ventricular cavities and thus it may fail to record the increased negative potentials of the ventricular cavities in the healed stages; nevertheless the anterior praecordial leads, being more advantageously placed, may record increased S waves (cases 32, 37, 38). Similarly it is unusual for lead VL to show increased height of its R wave although this feature may be present in the apical and axillary leads, the general pattern of which may point to left ventricular dilatation or hypertrophy. However if there is extensive scar tissue in the posterior ventricular wall lead VL along with leads V3 to V6 may be freed from the opposing potentials normally generated in the posterior wall and thus all five leads may display a late increase in the height of their R waves. This is exemplified by the two cases showing signs indicative or suggestive of aneurysmal dilatation of /

/ of the posterior wall (Nos. 28, 32).

While the unipolar limb leads thus provide further information about posterior infarction apart from its actual presence, they do not give any indication of its surface area. The praecordial leads are more informative in so far as the axillary leads V5, V6 and V7 record directly the effects of any lateral extension of the posterior infarction. The anterior praecordial leads, on the other hand, face towards the endocardial surface of posterior infarcts and thus will be expected to show changes opposite to those in leads facing the epicardial surface, such as the oesophageal leads and also lead VF, when the heart is in intermediate to vertical position. There is, firstly, acute depression of the RS-T segment during the stage of injury. As far as lead V1 is concerned, this may simply mean depression to the iso-electric level from its slightly elevated normal position; leads V3 and V4 show the greatest degrees of depression, e.g. two to three millimetres in cases 27 and 41. The RS-T depression is followed by a return of the RS-T junction to the iso-electric line. At the same time, the R and the erect T waves become exaggerated in height, due to suppression of potentials normally emanating from the posterior wall and normally antagonistic to positive potentials arising in the anterior cardiac wall. The registration of these signs by the anterior praecordial leads requires that there shall be some degree of

/ of parallelism between the plane of the infarct, the anterior cardiac wall and the chest wall; the line of the electrodes, shall be, in part, diametrically opposed to the centre of the infarct and the musculature of the anterior cardiac wall shall be intact. In view of the number and frequently unassessable nature of these variables, it can only be premised that if reciprocal changes are present in several leads, a large infarct can be expected.

In spite of such qualifications, the reciprocal changes are informative in other ways. The manner of their disappearance is an index of the healing of the infarction. Case 27 shows, during the first few weeks after onset, progressive diminution in the height of the R waves of leads V1 and V2, the former especially, indicating an early re-establishment of electrical forces normally averse to the R waves of the anterior praecordial leads and independent of any subsequent changes in the S waves. As early as the end of the first week the T waves have become exaggerated. Such signs of early recovery at the edge of an infarction may have their physical substrate in the absorption of oedema or other early resolving process.

Similar prominent R waves which are unaccompanied by exaggeration of the T waves and which diminish as healing progresses in the later weeks are seen in the anterior praecordial leads of case 37, but the signs of healing are confined to these leads in this case. The tall R waves /

/ tall R waves which are to be expected in the later stages of healing in leads V4 and V5 do not occur owing to their proximity to the lateral part of the infarction. Similar signs of healing, although temporary, are seen in case 28 where, four months after onset, lead V1 has become normal again in so far as previously high R and T waves have both diminished in size; there are slight changes of a similar kind in lead V2.

Prominent R waves in the anterior praecordial leads may be due to causes other than the reciprocal effects of infarction, especially if they do not diminish in height at the stage of healing when their associated T and S waves have evolved in the usual manner. Such an instance is provided by case 33 where, as suggested in the text, the prominent R waves may be due to right ventricular hypertrophy.

While the reciprocal changes are of the same nature in every case, they are superimposed on the pre-existing pattern of the anterior praecordial leads which varies greatly from case to case because of the vagaries of the electrical position of the heart. In vertical and semi-vertical hearts the anterior praecordial leads normally register right ventricular and "cavity" potentials, as seen by their small R and long S waves. Because of the placing of the electrode in relation to the vertical position of the heart, it may not be opposed, in the V1,

/ the V1, V2 and V3 positions, to small posterior infarctions and hence these leads may not be influenced by the electrical changes caused by the infarction. They may therefore show no exaggeration of their R and T waves, but this fact does not preclude them from sharing in the increased negative potentials of the left ventricular cavity which are a feature of the later stages of healing and which are demonstrated by tall R waves in the apical and axillary leads and deep S waves in the anterior praecordial leads. Case 34 is an example of the anterior praecordial leads remaining unaffected until the later stages of healing and compensation, and in case 40 also, the only sign throughout, apart from those due to temporary septal spread, is late elongation of the S waves.

It is not uncommon in vertical or semivertical hearts for the electrode in the anterior praecordial positions to be in opposition to the zone of ischaemia and not to the zone of necrosis and hence high peaking of the T waves occurs as the sole reciprocal sign in the earlier weeks, although there is no increase in the R waves. These features are followed by elongation of the S waves as above described (cases 44, 45 and 46). Associated with these long S waves in leads V1, V2 and V3 there may or may not be specially tall R waves in leads V4, V5 and V6. This depends on the degree of clockwise rotation of the vertical or semivertical heart. If it is of greater degree, then /

/ then the tall R waves do not appear since the left ventricle is less directly accessible to the exploring electrode (cases 34 and 40). If it is of lesser degree, leads V4, V5 and V6 record exclusively left ventricular potentials and tall R waves occur in these leads (cases 43, 44 and 46). A picture similar to the latter may also be found in the healed stages in intermediate and semihorizontal hearts, viz. long S waves in the anterior praecordial leads and tall R waves in the apical and anterior axillary leads (case 30).

A different electrocardiographic pattern in the healing of infarction is provided by horizontal or semihorizontal hearts with counterclockwise rotation. In this position of the heart the normal pattern in the anterior praecordial leads shows small S waves and prominent R and T waves. This is a left ventricular pattern and is registered by these leads because the counterclockwise rotation of the heart causes the left, and not the right, ventricle to be presented to the exploring electrode in the V1, V2 and V3 positions. When such a heart becomes the seat of posterior infarction, there may be a further increment in the size of the R and T waves, or of the T waves alone, if the anterior praecordial positions are in opposition only to the zone of ischaemia. It may be necessary to study the subsequent evolution of the RST formation in order to evaluate retrospectively the relative influence of cardiac position /

/ position on the one hand and the reciprocal effects of infarction on the other in the shaping of the electrocardiographic pattern of leads V1, V2 and V3 in the early weeks after infarction. Five cases of the present series have horizontal or semihorizontal hearts with frank counterclock-wise rotation around the long axis of the heart and hence the early anterior praecordial leads of all five cases bear a close resemblance to one another (Nos. 31, 36, 38, 56 and 59). But these leads may take little cognisance of rotation around other axes, e.g. rotation of the apex around a vertical axis and, furthermore, there is naturally much diversity in the relationship of the left and right arms to the posterior infarction. Hence there is little similarity in the unipolar and standard limb leads of the five cases.

Case 36, three and a half months after onset, is an example of persistently tall R and T waves and short S waves in leads V1, V2 and V3, probably therefore representing, in large measure, a basic left ventricular pattern, since changes due to very satisfactory healing are prominent in other leads, especially those changes activated by increased potentials in the left ventricular cavity. In case 31 on the other hand, S waves re-appear in leads V1 and V2 with slight increase in the R waves, which, when considered with the increased height of the R waves in the other praecordial leads, are probably an expression of the enhanced left ventricular function of the healed stages of posterior /

/ posterior infarction. In case 38 - also a horizontal heart with some counterclockwise rotation - both R and S waves show considerable increase in amplitude in the late stages of healing. As discussed in the text, these R waves are the result of increased left ventricular function on the one hand and of inert scar tissue in the posterolateral wall on the other.

The same pattern may be reached in the praecordial leads in hearts which are not demonstrably horizontal or counterclockwise rotated in the first instance, and it is a fair presumption that the same dual explanation holds good. The case of ventricular aneurysm (No. 28) shows very large R and S waves in the anterior as well as the other praecordial leads and in case 32, which also has prominent R and S waves, there is probably considerable scar tissue at the site of infarction, as explained in the text.

Lastly, reciprocal exaggeration of the T waves does not occur in the presence of pericarditis. In cases 49 and 55 pericardial friction was heard over the praecordium and in both cases the T waves of the anterior praecordial leads are of normal size, bifid or low.

While, therefore, evidence of the size of a posterior infarct is rather indirect there are two regions, viz. the septum and the lateral wall of the left ventricle where extension of infarction may produce direct effects on the unipolar limb or praecordial leads in relation to them. As /

/ As previously discussed, involvement of the septum in a horizontal heart may be documented by a QS deflection in lead VF along with RS-T signs of infarction. Lead V1 is frequently in close relationship to the posterior part of the septum and faces its right side. Hence infarction in this region causes disappearance of the early R wave of lead V1 which is due to septal activation; a deep QS deflection remains, accompanied by elevation of the ST junction, upward convexity of the ST segment and later by inversion of the T wave. In addition lead VR may show elevation of its ST segment and, later, inversion of its T wave, but a QS deflection in this lead cannot be considered abnormal. Such a similarity of lead VR to lead V1 would indicate that it is also in relation to the septal infarction. Septal spread of infarction occurred while the patient was under observation, in case 40, as judged by the appearance of diagnostic signs in lead V1 and to a less extent, in leads V2 and V3. The new signs which occur in lead VR are also attributable to the same cause. In all four leads a small early R wave finally re-appears indicating re-establishment of septal function. In some vertical hearts lead VL as well as lead VR may face the right side of the septum and both may therefore record septal potentials. In case 34, a tiny R wave develops in lead VR and a small R wave increases in height in lead VL, four and a half days after onset, due to early restoration of septal function. Less conclusive /

/ Less conclusive evidence of septal involvement is provided by case 42, in which small Q waves appear de novo in the anterior praecordial leads, indicating suppression of the normal early activation of the left side of the septum. In addition the R waves are reduced, their diminution being due either to the disturbance of conduction in the left side of the septum or to temporary right ventricular strain, as discussed in the case report.

Involvement of the lateral wall by the necrotic zone of infarction can be diagnosed in three of the twenty-eight of posterior infarction so that the designation of posterolateral infarction can be applied (cases 36, 37 and 38). In case 36 there is a diagnostic QR complex with upward bowing of the ST segment and inversion of the T wave in lead V6 and, to a lesser degree, in lead V5, ten days after onset. In case 37, four weeks after onset, there is a small broad ventricular complex, with, however, a definite Q wave, an ST plateau and an inverted T wave in lead V7, whereas in lead V6 there are only equivocal ST-T changes. Case 38 is of later date, the tracing not being obtained until two months after onset. Nevertheless there is a diagnostic Q wave in lead V6, the R wave is short compared to that of lead V5 but, at this date, the ST-T segment is flat. In all three cases the infarction is clearly subendocardial. Not only do the axillary leads demonstrate the presence of infarction beneath them, but they also show

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/ show the process of resolution from the periphery of the infarct, i.e. the V5 level towards V6. Rapid and complete repair is demonstrated in case 36 where, at the end of three months, the axillary leads are practically normal. The outcome in case 37 is much less satisfactory where, at the end of six months, leads V6 and V7 are practically unchanged, indicating minimal healing. In the third example (case 38) the final picture in leads V6 and V7, fifteen months after onset is that of a typical conduction defect - a broad conspicuous Q wave, a delayed R wave and a nondescript ST-T formation.

Equivocal changes also occur at the V7 and V6 level. For example, case 58, ten and a half weeks after onset, shows a significant Q wave, a rather short R wave and a shallow diphasic T wave. The Q wave would indicate extension of the posterior infarction into the subendocardial layers of the posterolateral wall while the interval from onset of illness would account for the scantiness of the ST-T changes. Not infrequently the axillary leads exhibit slight elevation or upward convexity of the RS-T segments with flat or inverted T waves without the presence of significant Q waves (cases 33, 39, 48 and 59). As a rule the associated R wave is short and an S wave is present - a pattern which is found where the transition from a right to a left ventricular pattern is well to the left in the praecordial series of tracings. It suggests some clockwise /

/ clockwise rotation of the heart around its own long axis or backward shift of the apex around a vertical axis. The ST-T pattern is very suggestive of underlying aschaemia such as surrounds the central zone of infarction. With healing the T waves become upright or at least flat. In other instances the T waves of the axillary leads are flat even in early tracings and as healing advances, they become upright again (cases 34, 44 and 45).

Besides indicating whether or not the lateral ventricular wall is involved by extension from a posterior infarction, the axillary leads, especially V7, provide evidence of residual lesions, the commonest of which is a conduction defect in the posterolateral wall. This fault may follow not only posterolateral infarction, as described above, but also plain posterior infarction where the original axillary leads have demonstrated, at most, ischaemia of the lateral wall (cases 33, 34, 39 and 45). The process would appear to be a late spreading and anchoring fibrosis around the original site of infarction, sufficient, in some cases to interfere with local conductivity. The signs of a local conduction defect are well known: viz. delay in attaining the peak of the R wave, which is low and associated, as a rule, with a flat or shallow inverted T wave. The presence of a significant Q wave is not essential for the diagnosis of a conduction defect, but it is an indication that the subendocardial /

/ subendocardial layer of muscle has been involved. The axillary leads may supply evidence of subendocardial ischaemia, i.e. the ST segments, though bowed upwards, shelve downwards to diphasic or inverted T waves (case 35). It may be widespread, as in the case cited, where leads V4 to V7 inclusive are affected. Lead VL and the reciprocal of lead VR have a similar configuration and hence also the shelving ST and diphasic T are transferred to standard limb lead I. Similar subendocardial ischaemia is seen in the axillary leads of case 42, where, after the depression reciprocal to posterior infarction has passed off, there remain straight ST segments, shelving downwards to inverted T waves. However the final pattern in this case cannot be differentiated from that of left ventricular hypertrophy or dilatation.

Lastly the apical and axillary leads provide evidence of the functional recovery and compensation achieved by the left ventricle. Attention has been drawn to the lengthening of the S waves in the anterior praecordial leads as an indication of increased potentials in the left ventricular cavity. If the heart is vertical or semi--vertical with lesser degrees of clockwise rotation, these long S waves are accompanied by tall R waves in the apical and axillary leads. If these features represent moderate increases in the depth and height of the respective waves when comparison is made with previous tracings, then it is

/ is reasonable to suppose that they represent increased functional activity on the part of the left ventricle (cases 44 and 46). If there is, in addition, slight sagging of the ST segment in the axillary leads, then the possibility of early left ventricular hypertrophy arises (case 43). If, however, there is a greater degree of clockwise rotation of the vertical heart, then there is no significant change in the height of the R waves in the apical and axillary leads, the sole difference being the increase of the S waves in the anterior leads, as previously described (cases 34, 40 and 50).

Similar considerations apply to intermediate, semihorizontal and horizontal hearts. Case 30 presents a closely similar picture, viz. long S waves in leads V2 and V3 and tall R waves in leads V4 and V5 but the end result is still within normal limits. Nevertheless, comparison with the previous tracing where the S and R waves are only of average length raises the possibility of increased functional activity of the left ventricle. Furthermore, unlike the cases just cited, this infarction affected more than the subendocardial layer. Fibrotic foci remain in the posterior wall, sufficient to cause a local conduction defect, as seen in lead VF, and to cause abnormal standard limb leads. Hence the possibility must be considered that this scar tissue, being inert electrically and hence eliminating potentials usually opposite to the positive waves of the praecordial leads, may be responsible for /

/ for passive exaggeration of the R waves in leads V4 and V5, although probably the increments are not great enough to warrant this view. Case 31 is similar, but in addition, there is some counterclockwise rotation of this horizontal heart. Hence, from the level of lead V2 onwards, the RST pattern is left ventricular in origin and the R waves of leads V2 and V3 and even of lead V1 share in the moderate increase in height seen in those of leads V4 and V5. Comparison with the previous tracing shows that the S waves of the anterior praecordial leads have been resuscitated, as pointed out in a previous section. The entire picture is one of increased left ventricular function and, as in the previous case, there is no clue to the presence of scar tissue in the posterior wall, which is responsible for the large Q waves of leads II, III and VF. Cases 29 and 41 are also of this type.

Large S and R waves characterise the late tracings in case 32 but there are several additional features. The actual increments of the S and R waves are striking when comparison is made with the previous tracing; the R waves of the anterior as well as the apical and axillary leads have increased and there is a conduction defect in the lateral wall of the left ventricle, as seen in leads V6 and V7. These facts would suggest that while there is increased functional activity there is also a fair extent of scar tissue in the posterior ventricular wall - a fact which /

/ which is indicated, in a general way, by the standard limb leads and lead VF. The same analysis can be applied to case 28, where there is a posterior cardiac aneurysm. There is no doubt in this case that all the late praecordial leads are frankly abnormal in their very tall and very deep deflections and hence it is less surprising to find the fixed picture of posterior infarct in the standard limb leads and lead VF. The foregoing cases illustrate that while the diagnosis of a healed posterior infarction may not even be inferred from the praecordial leads, yet they contribute to the assessment of the nature and extent of the posterior lesion, diagnosed by the standard limb leads and lead VF. Case 45 illustrates the same point for, in addition to elongation of the S waves in leads V1, V2 and V3 and greatly heightened R waves in leads V4 and V5, there is also broadening and splintering of the QRS deflection with sagging of the ST segment and diphasic T waves in the axillary leads. Thus there is, in addition to compensatory functional activity and scar tissue in the posterior wall, a severe conduction defect in the lateral wall. While the standard limb leads and lead VF indicate the presence of gross pathological change locally, the praecordial leads elucidate the degree of compensation and of persistent abnormality.

SUMMARY

Twenty-eight cases of posterior infarction have been analysed in respect of their unipolar limb and praecordial leads. Six show a QS deflection in lead VF; in one of the six it can be ascribed to posterior cardiac aneurysm. In the remaining five cases, the means of differentiating a QS deflection due to horizontal cardiac position from those due to infarction, either of the posterior part of the septum in a horizontal heart or of the posterior wall in other cardiac positions, are discussed; the best differential feature in favour of infarction, especially of the posterior wall, is the subsequent reappearance of an R wave.

Significant QR deflections in lead VF are displayed by the original tracings of eleven cases. Whether it is present ab initio or whether it develops out of a previous QS deflection, this pattern along with inversion of the T wave frequently becomes fixed or modified into that of a conduction defect.

QRS deflections in lead VF, which are smaller than five millimetres, are recorded in the original tracings of seven cases and develop in other four cases while under observation. Hence standard limb leads II and III must depend largely on leads VR and VL, respectively, for the /

/ the configuration of their QRS deflections and hence it is important to assess in what measure leads VR and VL are affected by posterior infarction. This evaluation is also applicable even if the QRS of lead VF is not specially small.

Lead VR may cause exaggeration of the Q wave of standard limb lead II if it shows a normal small r wave. Lead VR largely supplies the R wave of lead II but the presence of posterior infarction prevents it from being tall because of the scanty contribution from lead VF. Lead VR shows the specific effects of septal extension of infarction in two cases of the series. Otherwise posterior infarction affects only the RS-T deflection of lead VR in the same fashion as does anterior infarction, viz. by downward convexity of the RS-T segment and subsequently by upright peaking of the T wave.

Lead VL in nine out of twenty-eight cases presents a specific pattern, viz. absence of a Q wave, a sudden steep ascent of the R wave with early onset of intrinsic deflection and notching near the base of the descending limb. The importance of absence of the Q wave in standard limb lead I in the diagnosis of posterior infarction is discussed. There are only three instances in the present series where a Q wave appears in lead I - a circumstance to be explained by involvement of the lateral ventricular wall.

The signs shown by the anterior praecordial leads in /

/ in the presence of posterior infarction are reviewed. The ultimate pattern depends on cardiac electrical position and on the spatial relationship of the electrode in the V positions and the infarct. Early sequential changes may provide an indication of local healing, e.g. diminution in size of a previously exaggerated R wave in leads V1 and V2. Signs which are attributable to spread of infarction into the septum may occur in the anterior praecordial leads, viz. abolition of a previous R wave with appropriate RS-T signs or the development of a Q wave.

The axillary leads may indicate involvement of the lateral wall in the infarction. In three of the twenty--eight cases it is involved in the central zone but there are several other cases where it is only implicated by the surrounding zone of ischaemia. The axillary leads also record the residual lesions in the lateral wall, commonly a conduction defect; less frequently, persistent subendocardial ischaemia.

The praecordial leads provide distinctive patterns of the healed stages of posterior infarction. In vertical and semivertical hearts with lesser degrees of clockwise rotation, there are long S waves in the anterior praecordial leads and tall R waves in the apical and axillary leads. If there is greater clockwise rotation, the axillary leads /

/ leads may not show tall R waves. Intermediate to horizontal hearts may show similar long S and tall R waves but if there is severe counterclockwise rotation of a horizontal heart, the R waves may be prominent from the V2 position leftwards. These signs suggest increased left ventricular function. If, however, there is considerable scarring in the posterior wall as documented by a fixed pattern of infarction in lead VF and a conduction defect in leads V6 and V7, there is further exaggeration of the R waves of the praecordial leads due to permanent failure of opposing forces.

SECTION VI

ANTEROPOSTERIOR INFARCTION

SECTION VI

ANTEROPOSTERIOR INFARCTION

Anteroposterior infarction is a more common pathological than clinical diagnosis. An infarction that involves both the anterior and posterior aspects of the apical walls is necessarily large and death may follow or a recent infarct may be found in the anterior wall of a heart already damaged by a previous infarct in the posterior wall or vice versa. On the other hand the clinical diagnosis, based on electrocardiograms may be uncertain. The classical pattern consists of abnormal Q waves in all three leads, accompanied early by elevation of the RS-T segment, greater in lead II than in leads I or III, and later by inversion of the T waves throughout but Myers et al (1949) found many variations from this pattern in their fifty-two cases of anteroposterior infarction, proved pathologically. They found that unipolar limb and praecordial leads were necessary for the localisation of this infarction. The praecordial leads portrayed more faithfully the anterior lesion than the left leg lead did the posterior. The causes of failure to diagnose the posterior continuation of the lesion were the same as those which operated when posterior infarction is present alone. Myers found that horizontal position of the heart was a/

/was a frequent cause of failure of diagnostic signs to appear in lead VF. Furthermore, irrespective of cardiac position, the continuance of the lesion from the apical one third of the posterior wall into its middle third greatly increased the positive findings in lead VF. However in the three cases of the present series, the diagnosis was entirely electrocardiographic and hence incontrovertible signs of infarction occur in lead VF.

Electrocardiographic diagnosis is further complicated by the effect of the one lesion on the other. Their relationship may act in the direction of militation or suppression of diagnostic signs if portions of infarcted wall are diametrically opposed to one another; this is more likely to arise with two infarctions of different date than with a single infarction. After all, an abnormal Q wave in any lead, upon which the diagnosis of infarction largely rests, depends on the integrity of the myocardium elsewhere, to generate negative potentials in the ventricular cavity which are registered as a Q wave by the electrode over the infarction. Thus signs of posterior infarction in lead VF may be modified by the presence of anterior infarction as exemplified by case 50. Similarly, the reciprocal effects of posterior infarction on the anterior leads may obliterate the signs due to antero-septal infarction.

Case 50 H. S., male, 52 years.

This patient was admitted in severe shock a few hours after the onset of pain in the lower chest. He had been in good health and had been walking to his work as a storeman in the morning after breakfast when he was suddenly seized with intense gripping pain in the lower sternal area. He was assisted to a nearby hut, but, as the pain did not abate, he was sent straightaway to hospital.

Pulse was 70 per minute, rising in the next few days to 100 per minute. Blood pressure was 134/90 on the day of admission but fell to 110/84 on the following day and 90/62 by the tenth day of illness. Thereafter it rose to 112/80 at which level it remained. The cardiac sounds were invariably soft. There was originally a leucocytosis of 14,000 cells per cmm. Radiologically the heart showed slight left ventricular prominence.

The patient made a good recovery and resumed work. When seen again three months after onset, he made no complaint of pain. Blood pressure was 126/90 and the cardiac sounds were of average intensity.

/ The first electrocardiogram obtained six hours after onset of pain shows elevation of the ST junction in standard limb lead 1 typical of anterior infarction. There is slight elevation of the junction in lead 2. There is no Q wave in lead 1 but a small Q is discernible in lead 2, and lead 3 shows a splintered QS deflection. At this early stage the T waves are upright in all three leads. Thus lead 1 and, to a lesser degree, lead 2 reveal evidence of recent anterior infarction, while lead 3 and also lead 2 show signs of posterior infarction of undefined date. The unipolar limb leads show that both the left and the right arm electrodes face the back of the heart as seen by the inversion of the P waves, the depression of the ST segments and the inverted T waves, of a shape typical of the intact ventricular wall opposite an infarction. Thus the elevation of the ST segment and subsequent inversion of the T wave of standard limb lead 1 are derived from the reciprocal of lead VR, mitigated by the pattern of lead VL. There is probably some backward rotation of the apex and thus the lower part of the infarction would readily be brought into relation with the diaphragm and the left leg electrode.

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In most of the serial tracings however lead VF displays a central not marginal zonal pattern so that it is probable the infarct actually involved the posterior part of the apex. The praecordial leads, six hours after onset, show severe elevation of the ST junction in leads V2, V3, V4 and V5; the ST segment is still concave upwards and the T wave upright. These findings are typical of the stage of injury - the subepicardial muscle has survived but is severely injured. A minute r wave is retained by lead V1 indicating maintenance of function in the anterior part of the septum. Deep QS deflections from the central zone of infarction are present in leads V2, V3 and V4, indicating transmural anteroseptal and apical infarction.

The second serial tracing taken one day after onset indicates some spread of infarction probably in the lateral wall as shown by the diminution in size of the R wave of lead V5. The physiological Q wave of lead VR has practically disappeared causing diminution in the already small R wave of standard limb lead 1.

At the same time this further reduction in the

negative potentials within the ventricular cavity has caused not only the virtual disappearance of the pathological Q wave from lead VF but also the resurgence of its R. wave. Thus the QRS complex of this lead has apparently reverted to a normal form albeit temporarily, in the face of spread of infarction in an opposing ventricular wall.

By six days after onset, elevation of the ST segment is decreasing in the praecordial leads and inversion of the end of the T wave is already conspicuous in the central praecordial leads. Lead VF shows a central zonal pattern which largely determines that of standard limb leads 2 and 3. These leads resemble one another because of the similarity of leads VR and VL. Thereafter the ST-T patterns of these leads diverge; that of lead VR shows the prominent upright T wave and slight depression of the ST segment found later over the unaffected ventricular walls opposite an infarction, whereas the ST-T segment of lead VL is of less defined type. Hence when lead VR in reciprocal is summated with lead VF to form lead 2 there is conspicuous inversion of the T wave but when combined with lead VL to form lead 1 the inversion is mitigated to a certain degree.

Subsequent serial tracings up to seven weeks after onset show the classical evolution of an anterolateral infarction in the praecordial leads. Lead V1 is practically normal as early as three weeks after onset. Deep QS deflections persist in leads V2, V3 and V4 and a small marginal pattern is seen in lead V5. Cove-plane T waves are present in leads V3, V4 and V5. The standard limb leads show inversion of T waves in leads 1 and 2 as before.

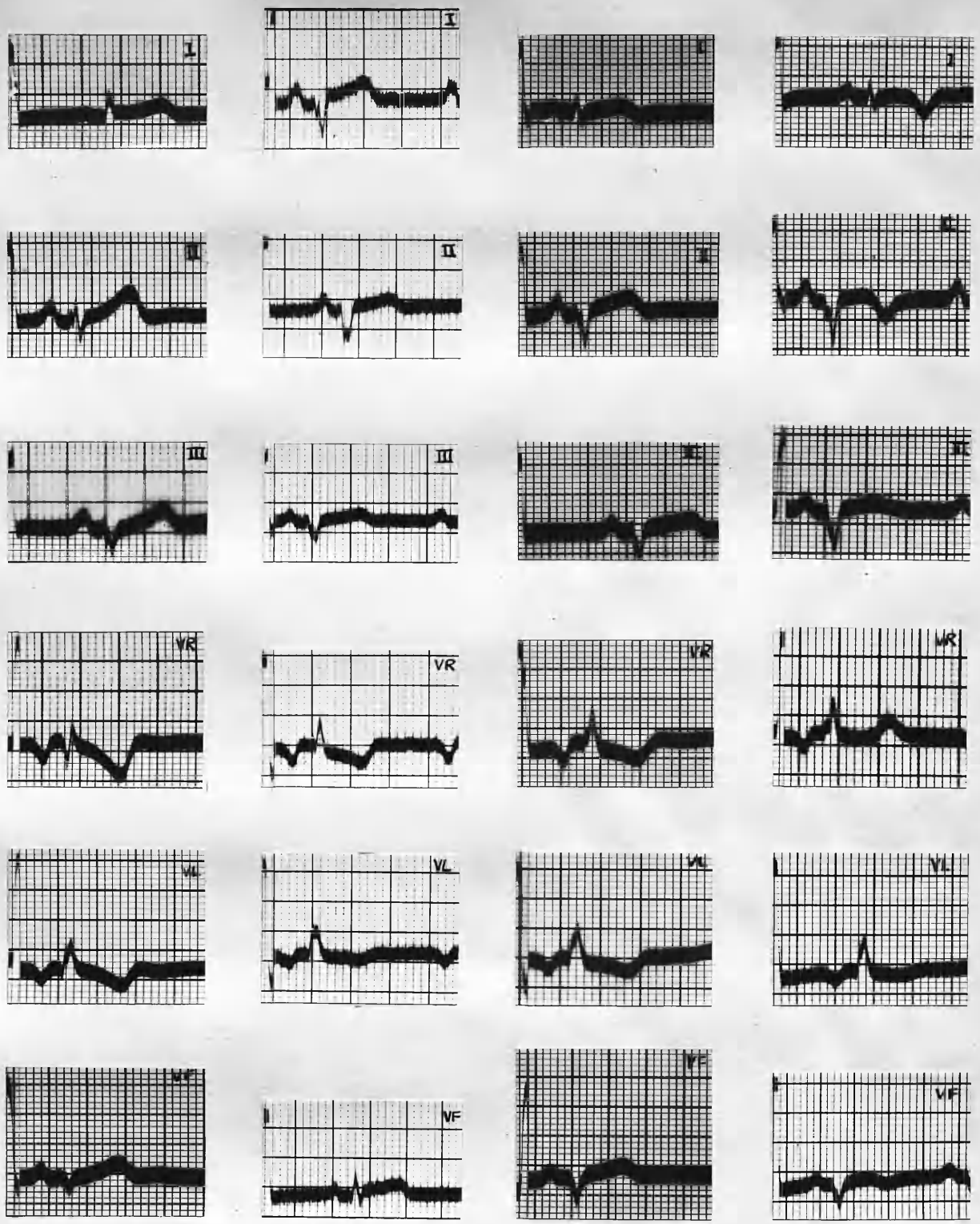
The final tracings taken almost three months after onset show, as before, left axial deviation. There is no Q wave in any of the standard limb leads and lead 1 alone shows shallow T inversion. Lead VR with its single tall R deflection is frankly abnormal. It represents potentials obtained from the posterior cardiac wall undiminished by those normally arising in the anterolateral cardiac wall and which normally precede as well as accompany the potentials of the posterobasal ventricular region. The left arm lead shows a tall slightly delayed R wave probably derived from the posterolateral ventricular wall which is more or less intact. Whether its height is enhanced by lack of antagonistic forces arising in the opposite wall similar to the relationship in lead VR is open

to question. The failure of tall R waves to appear in the axillary leads would favour the view that there was little, if any, functional increase in the lateral ventricular wall. Lead VF still shows a QS deflection rather deeper than those of previous tracings. Taken in conjunction with them, however, it probably still represents a central zonal pattern although as an isolated finding it may arise as a normal variant in a horizontal heart but this is unlikely to be the electrical position in this case. The praecordial leads show further signs of recovery over the right praecordium where the early R wave has increased in lead V1 and reappeared in lead V2. Deep QS deflections persist in leads V3 and V4. Recovery in lead V5 is seen by the resurgence of the R wave above the isoelectric level although its peak is delayed; presumably recovery is epicardial in situation.

This case is an example of a severe anterolateral infarct with involvement of the posterior aspect of the cardiac apex. The praecordial leads are typical of the former. The position of the heart involves backward rotation of the apex. The pattern of the unipolar limb leads is therefore somewhat unusual;

lead VF and not VL shows signs directly derived from the infarction.

Hence the unusual distribution of signs in the standard limb leads. Recovery at the right margin of the infarction is very satisfactory but there is non-functioning scar tissue at the previous central zone of infarction.



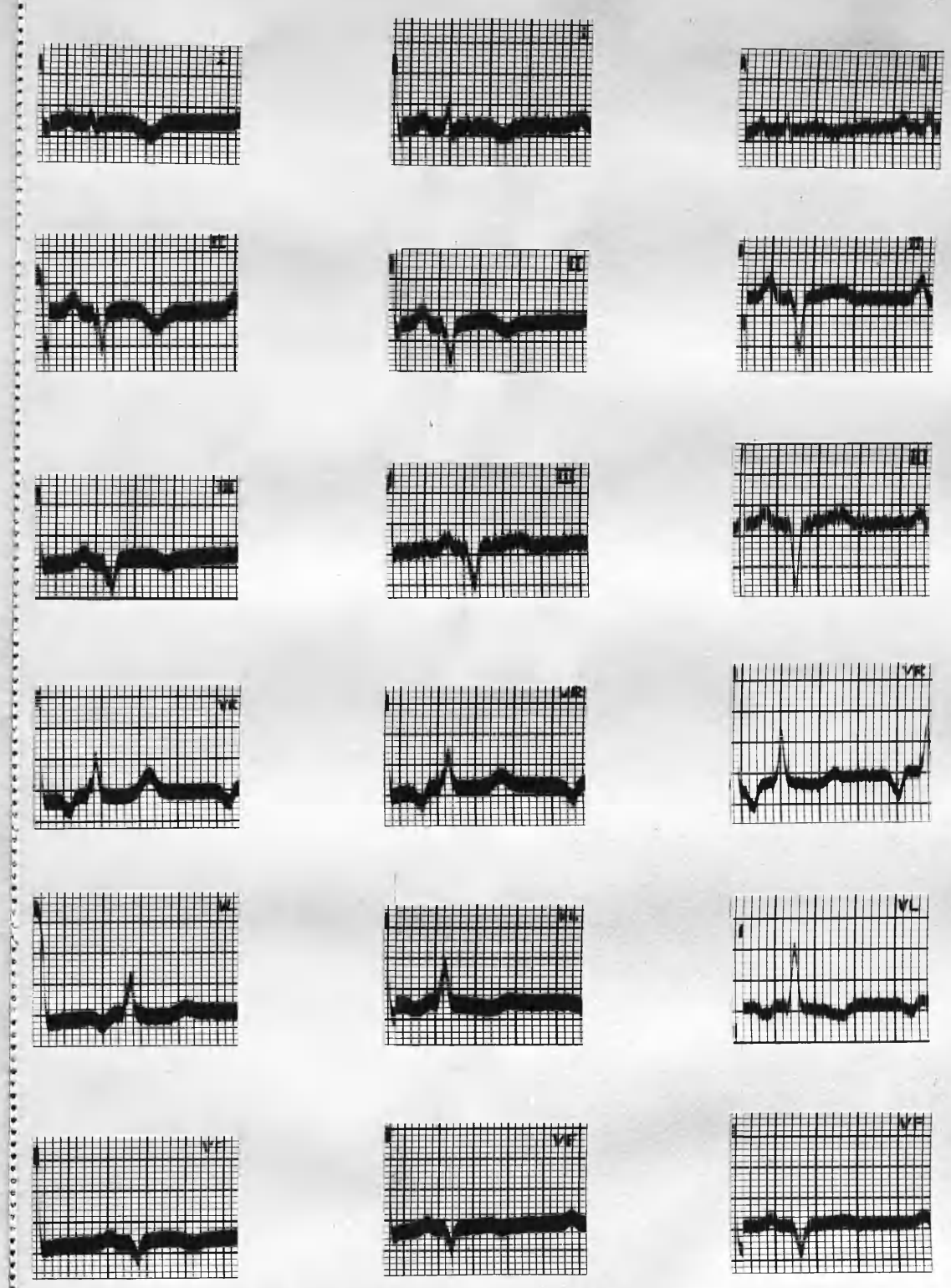
CASE 50, H.S. MALE, 52 YEARS.

6 HOURS AFTER ONSET.

1 DAY AFTER ONSET.

6 DAYS AFTER ONSET.

21 DAYS AFTER ONSET.

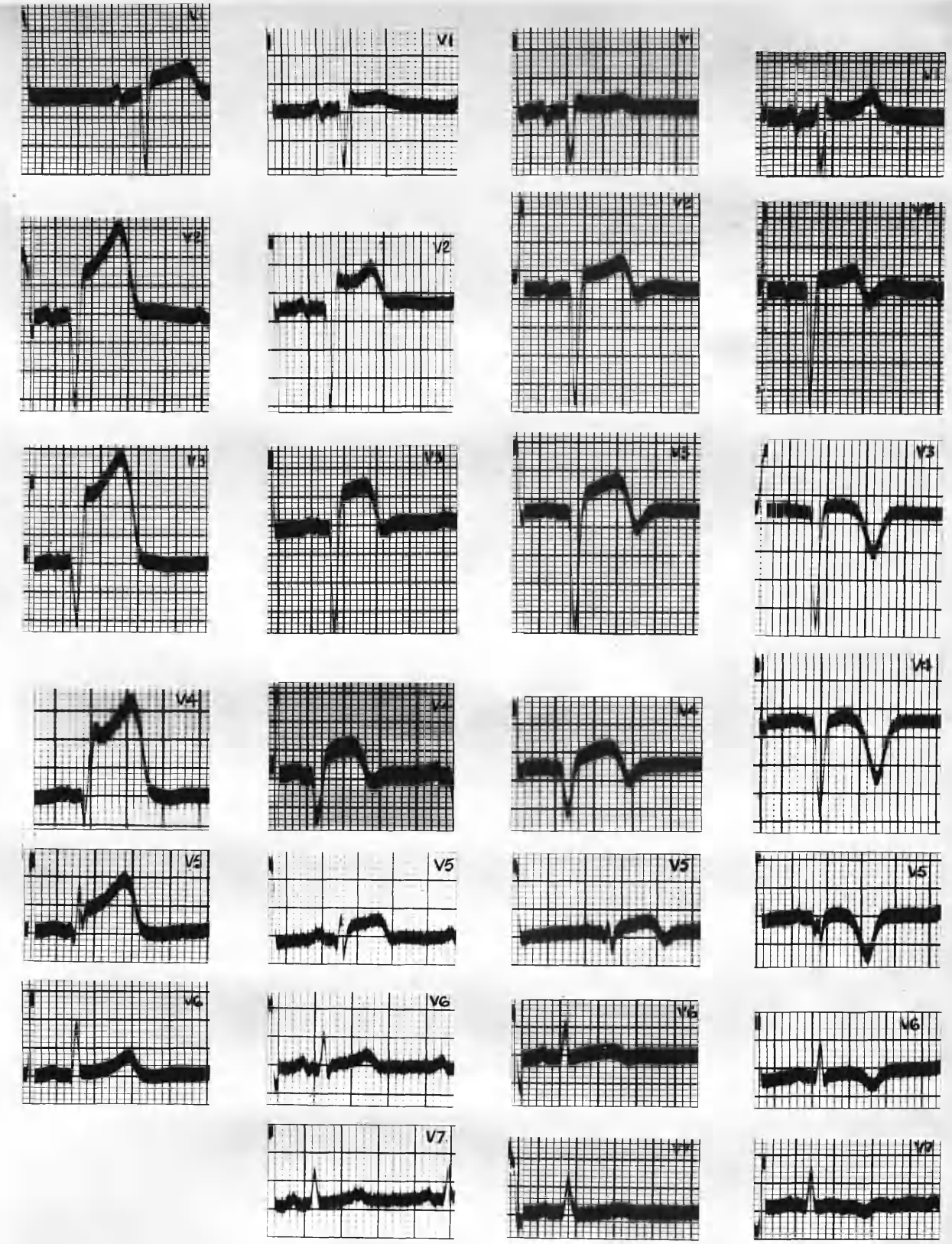


CASE 50, H.S. MALE, 52 YEARS.

ALMOST 5 WKS AFTER ONSET.

7 WEEKS AFTER ONSET.

ALMOST 3 MTHS AFTER ONSET.



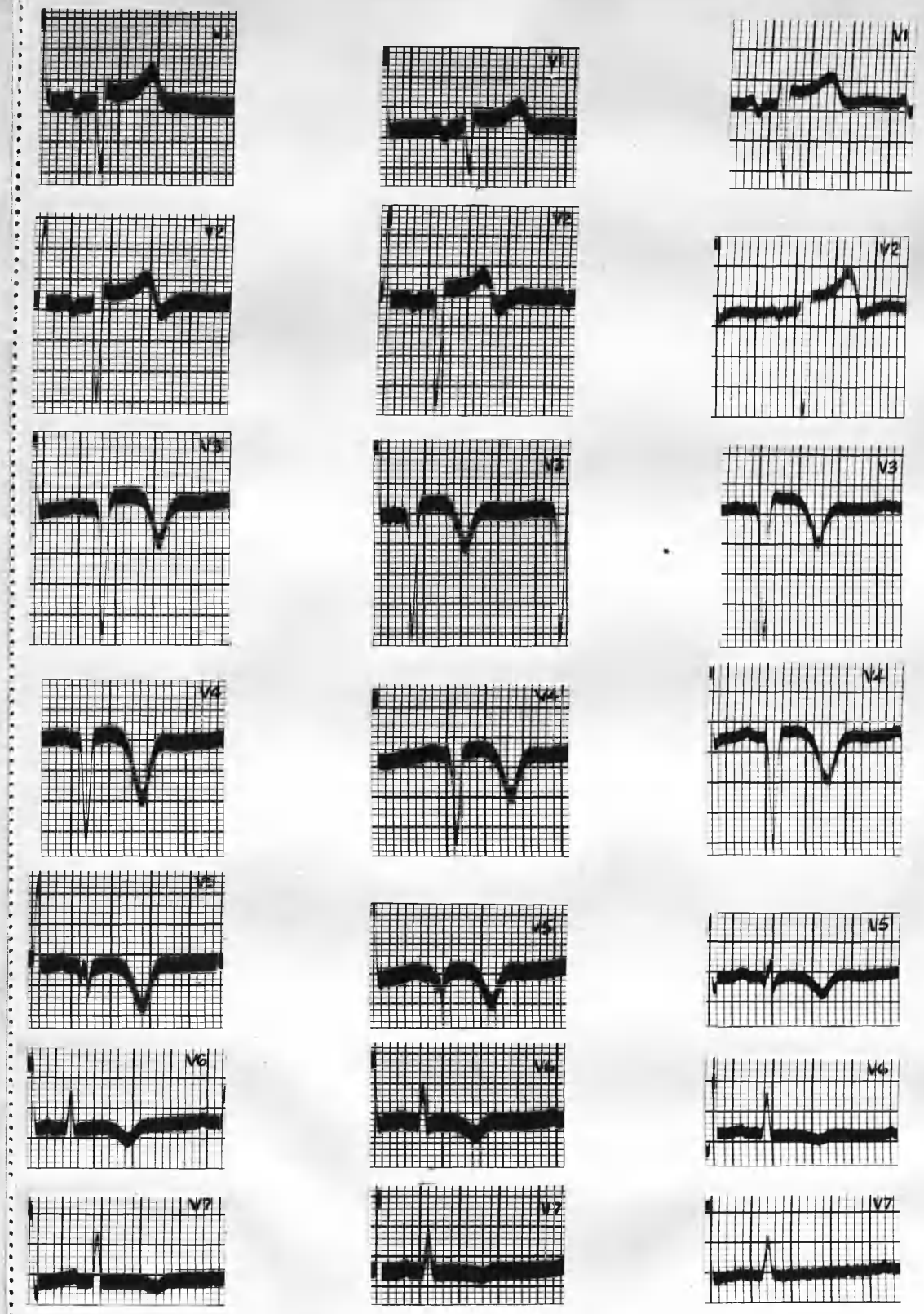
CASE 50, H.S. MALE, 52 YEARS.

6 HOURS AFTER ONSET.

1 DAY AFTER ONSET.

6 DAYS AFTER ONSET.

21 DAYS AFTER ONSET.



CASE 50, H.S. MALE, 52 YEARS.

ALMOST 5 WKS AFTER ONSET.

7 WEEKS AFTER ONSET.

ALMOST 3 MTHS AFTER ONSET.

Case 51, J.D., male, 36 years.

This patient had an attack of substernal pain lasting one hour and recurring frequently for twelve hours three weeks before the first electrocardiogram was obtained. When seen at the Out-Patient Department, blood pressure was 120/70; there was no cardiac enlargement clinically and the cardiac sounds were of fair quality.

Blood pressure remained at above level throughout the period of observation. When examined fifteen months after onset the cardiac sounds were loud; there was sharp pulsation over the praecordium generally but no clinical cardiac enlargement. The patient was free of symptoms but had not resumed his former employment of insurance agent; he had started work as a shop assistant.

The standard limb leads taken three weeks after onset of illness would at once raise the possibility of a combined anterior and posterior infarction. There is no R wave in lead 1, only a splintered QS deflection and there is a prominent Q wave in the small ventricular deflection of lead 2;

/

/ in all three leads however there is slight elevation of the ST take-off and slight upward convexity of the ST segments, best seen in lead 2. The possibility of a localised anterior lesion with general pericarditis must also be considered. The QRS deflections of unipolar limb leads VR and VF are so small that they are of limited value, but a prominent upright T wave is clearly seen in lead VR - a sign which is commonly found in this lead when it apposes an extensive recent anterolateral infarction. Lead VF shows upward convexity of its ST segment suggestive of an origin directly from infarction of the posterior wall. The deep QS deflection of lead VL may be derived from the central zone of infarction but the possibility of vertical cardiac position as its cause must be considered. The evolution of the lead in subsequent tracings leaves no doubt that the former is the correct interpretation.

The praecordial leads show a normal rS deflection in lead V1; thereafter in leads V2 to V6 inclusive, there is a deep QS deflection, typical of an extensive anterolateral infarction. The RS-T junction is elevated but for the anterior praecordial leads this is not greater than normal. Furthermore

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/ Furthermore these leads show peaked upright T waves. While a picture of this type is not infrequent in an old healed anterior infarction there is no history to suggest the previous occurrence of any infarction. The presence of an acute posterior lesion in this case which is clearly established by lead VF, has probably prevented the RS-T junction from rising to abnormal levels as it does in acute anterior lesions, and has caused the sharply peaked symmetrical T waves as reciprocal features. Leads V5 and V6 show along with deep QS deflections, the RS-T formation typical of the stage of injury with early organisation. There is elevation of the RS-T junction, upward convexity of the ST segments and shallow inversion of the T waves. A QS pattern is rare in leads V5 and V6 when anterolateral infarction is the sole lesion. Of twenty-four cases of anterolateral infarction where the infarction of the lateral wall was transmural, Myers (1949) found a QS deflection in both leads in only three and in lead V5 alone in nine cases. The rarity of a QS complex in lead V6 he explains on the grounds that the transmural lesion rarely occupies more than the apical one-third of the lateral wall and that uninfarcted portions of the lateral wall give rise to an R wave following the initial downstroke.

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/ While this may be so in the present case, backward rotation of the apex, the heart being in an intermediate to semi-horizontal position, would favour the transmission of impulses from the central zone of infarction to the axillary wall.

Lead VF six weeks from onset indicates clearly that the posterior wall is involved; although the QRS remains small, the Q wave is as large as the following R wave, there is elevation of the RS-T take-off, upward convexity of the ST segment and early inversion of the T wave. The pattern of lead VR is typical of that lead when it apposes a large recent anterior infarction and at the same time is in relation to the back of the heart. The inverted P wave is common in leads derived from this region of the heart. The absence of an early negative ventricular wave which is normally derived from regions of the heart activated before the posterobasal wall indicates fairly extensive damage to ventricular muscle. The delayed positive deflection is due to activation of muscle of the posterobasal cardiac wall subtended by the electrode on the right arm and undiminished by any opposing forces. The

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/ The depression of the ST segment and upright peaked T wave are typical of a lead facing the endocardial surface of an infarction.

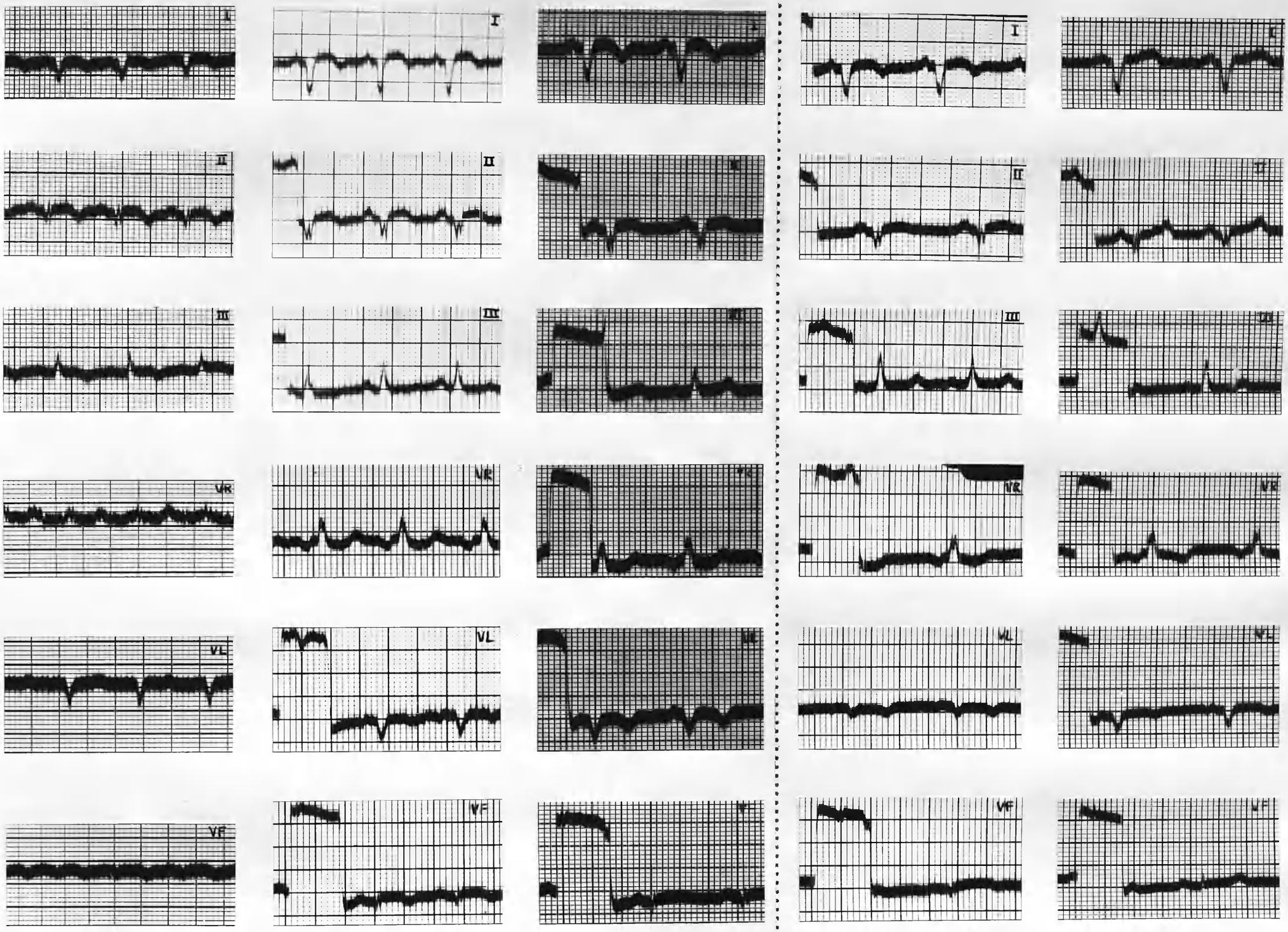
In the later tracings the chief sequential changes are seen in those leads which reflect changes in the posterolateral part of the infarction, Viz. in leads V6 and V7. Lead V7 shows progressive diminution in the size of its Q wave and increase in its R wave. Its ST segment is isoelectric and its T wave upright in tracings obtained fifteen months after onset. Similar regenerative changes are seen in lead V6 where a positive R wave finally re-appears but the Q wave persists although considerably shortened. As in lead V7 the S-T contour approaches normal.

There is no evidence of such recovery of function in the anterior praecordial leads. The chief sign is the reversion to the usual asymmetrical form of T wave with the RS-T junction remaining slightly above the base-line as in a normal tracing. There are persistent deep QS deflections in leads V2, V3, V4 and V5 which conform to the usual pattern of healed anterolateral infarction.

A correspondingly sharp distinction can be drawn /

/ drawn in the unipolar limb leads. Lead VF returns to normal indicating a satisfactory degree of recovery in the posterior part of the infarction whereas leads VR and VL show practically no change in the early phase of their ventricular complex which remains grossly abnormal in each case and which is derived from the poorly healed anterior infarction. The ST-T segments have reverted to the isoelectric level. The QRS pattern of the standard limb leads is largely governed by that of leads VR and VL because the QRS complex of lead VF is very small partly because of the intermediate to semi-horizontal position of the heart. Hence their signs are derived from the poorly healed anterior infarction and do not indicate the posterior lesion.

This case presents the signs of an extensive anterolateral infarction with involvement of the posterior part of the apex. This extension shows satisfactory repair but the anterior lesion shows no regenerative sign.



CASE 51, J.D. MALE, 36 YEARS.

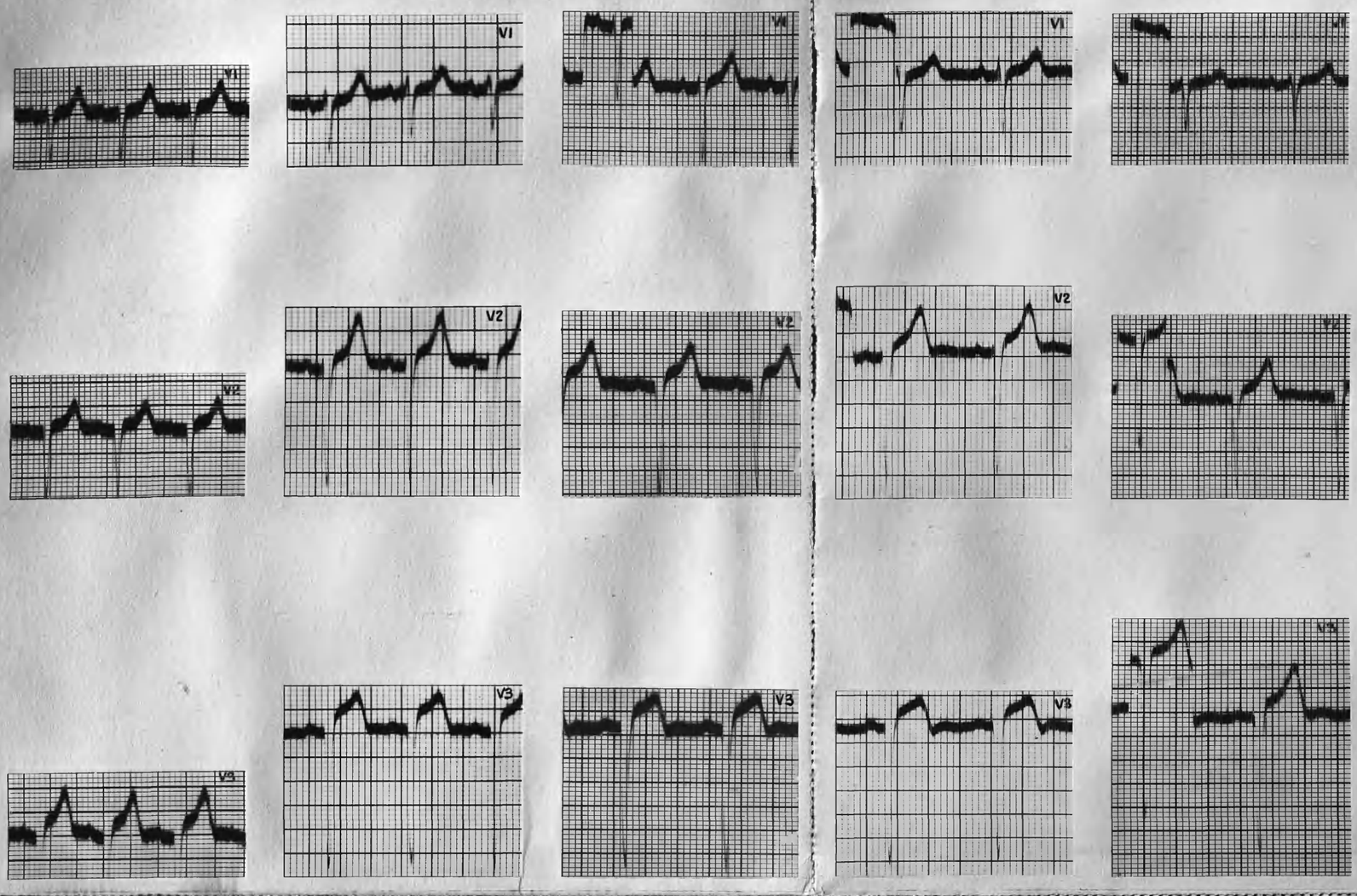
3 WEEKS AFTER ONSET.

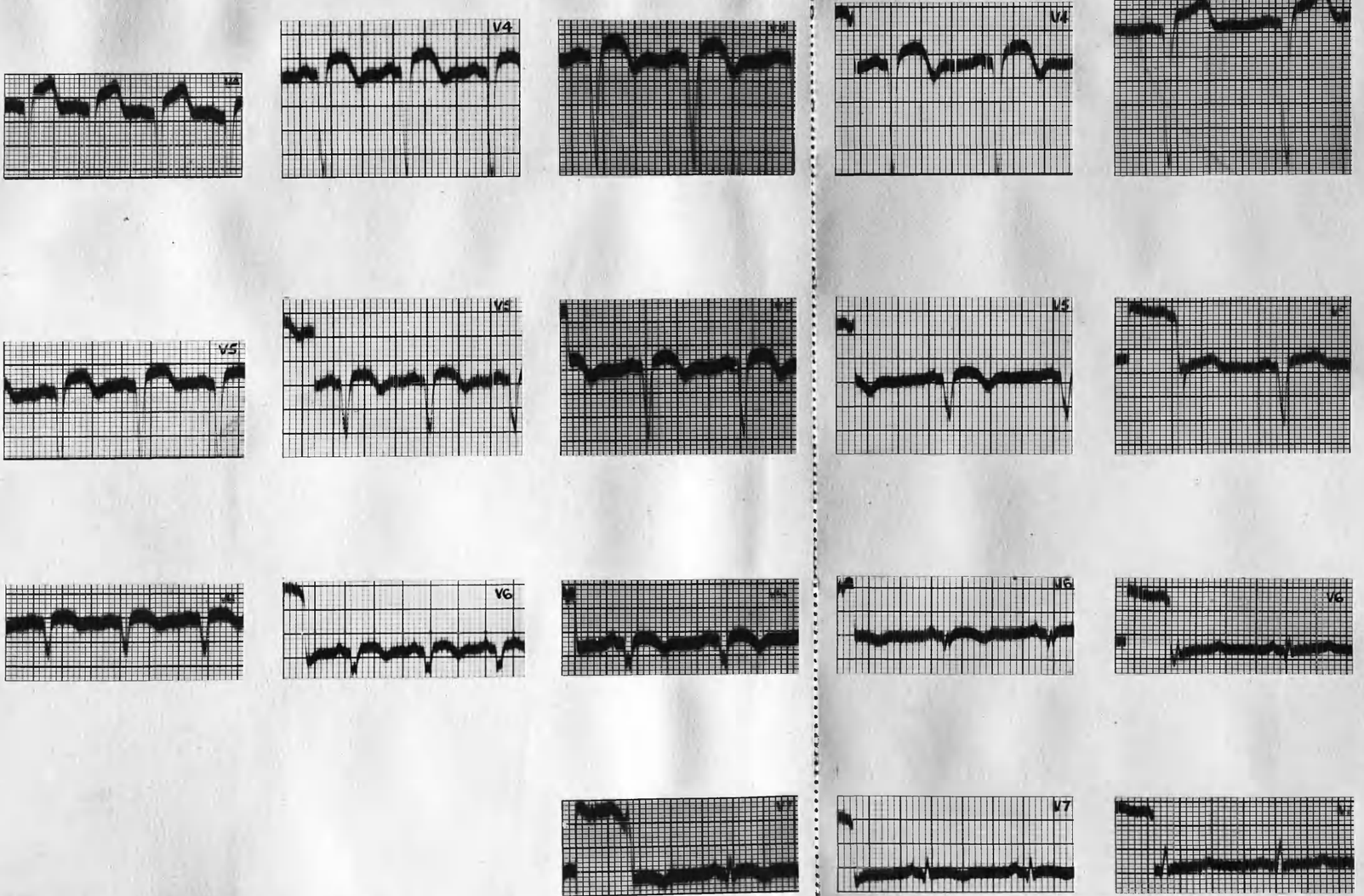
6 WEEKS AFTER ONSET.

9 WEEKS AFTER ONSET.

11 WEEKS AFTER ONSET.

15 MONTHS AFTER ONSET.





CASE 51, J.D. MALE, 36 YEARS.

3 WEEKS AFTER ONSET.

6 WEEKS AFTER ONSET.

9 WEEKS AFTER ONSET.

11 WEEKS AFTER ONSET.

15 MONTHS AFTER ONSET.

Case 52, A.R., male, 46 years.

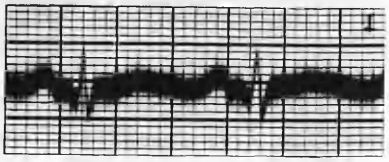
This patient had been treated elsewhere for an attack of myocardial infarction which occurred fifteen months before he attended the Out Patient Department. He was then complaining of dyspnoea on exertion and cough. He was of stocky build and the chest was very broad. There was slight cyanosis of face and lips. Blood pressure was 134/100. The cardiac sounds were of very poor quality. When seen again a year later there was little change in his condition. Blood pressure 150/90. The cardiac sounds were soft. Slight cyanosis persisted. Radiologically there was marked enlargement of the transverse diameter of the heart which showed a hypertensive configuration; the lung fields showed early passive congestion.

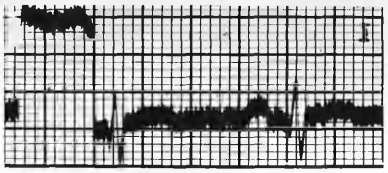
An electrocardiogram taken at his first attendance shows diagnostic signs of posterior infarction in standard limb leads II and III. There are conspicuous Q waves; the ST segments show upward convexity and the T waves are inverted. In view of the interval elapsing from the original attack of myocardial infarction, it is probable that these features represent a fixed pattern. Standard limb lead I is also abnormal. There /

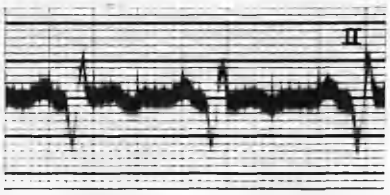
/ There is a borderline Q wave and the T wave is very low; these signs would also raise the possibility of a previous anterior infarction. The presence of a posterior infarction is confirmed by lead VF. Lead VL shows no significant feature but the early ventricular complex of lead VR is entirely positive which is a common finding in this lead in severe anterior infarction, the usual negative deflection of this lead being due to activation of muscle in the anterior ventricular wall. The praecordial leads show no frank abnormality in leads V1 and V2 indicating normal septal function but leads V3 and V4 show deep QS deflections with normal ST-T formations indicating a healed anterior infarction. Leads V5 and V6 are typical of a severe old-standing subendocardial infarction in the lateral wall. They are similar in form to lead VF and together indicate the subendocardial involvement of the lateral and posterior regions of the apex.

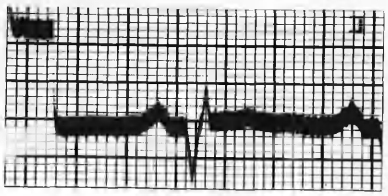
The passage of a year caused little change in the limb leads, standard and unipolar, except for flattening of the RS-T segments in leads II, III, VL and VF. However regenerative signs are apparent in the lateral part of the infarction and consist of increase in height of the late R waves in leads V5 and V6 indicating /

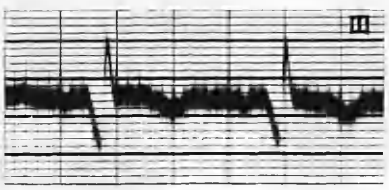
/ indicating some recovery of function in the subepicardial layers of the lateral ventricular wall. The anterior and apical leads show the persistent signs of a healed anteroseptal infarction. The absence of specific signs of the anterior infarction in standard limb lead I is due to their scantiness in lead VL which in turn has recorded the potentials of a largely unaffected part of the lateral wall - a transference facilitated by the transverse position and counterclockwise rotation of the heart.

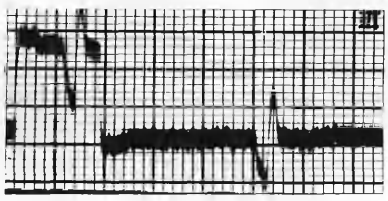


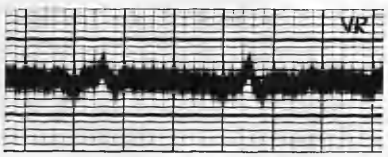


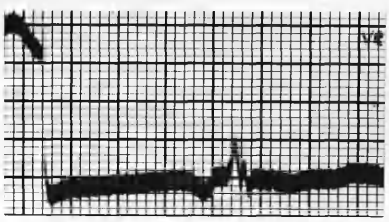


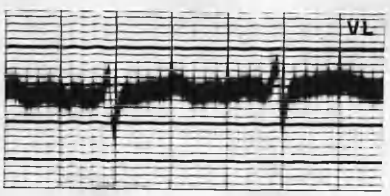


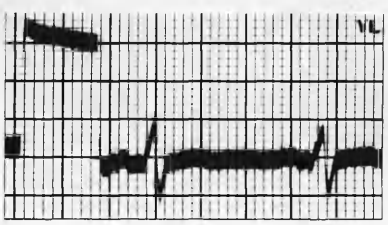


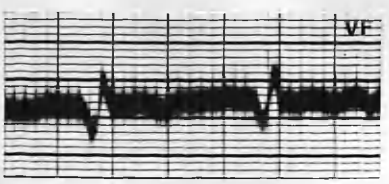


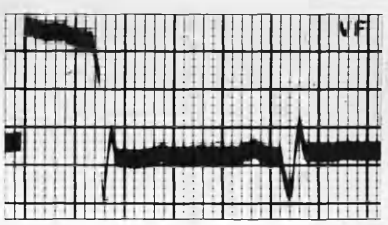








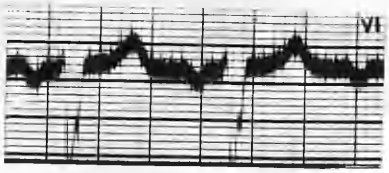


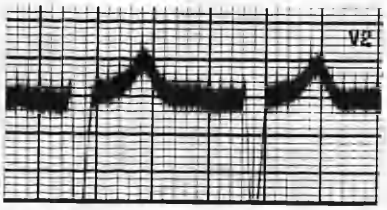


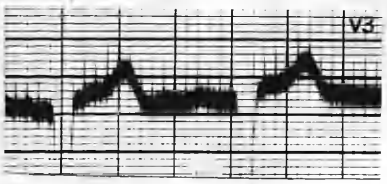
CASE 52, A.R. MALE, 47 YEARS.

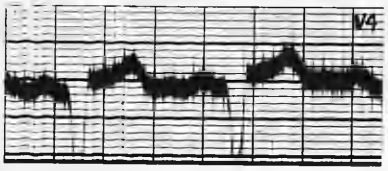
15 MONTHS
AFTER ONSET.

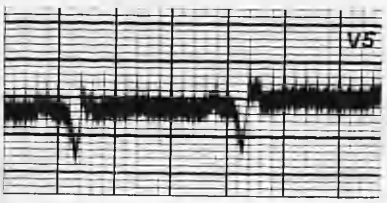
2¼ YEARS
AFTER ONSET.

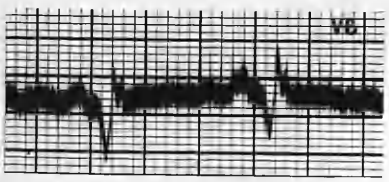




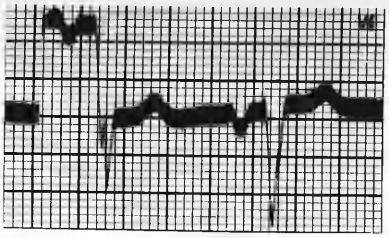


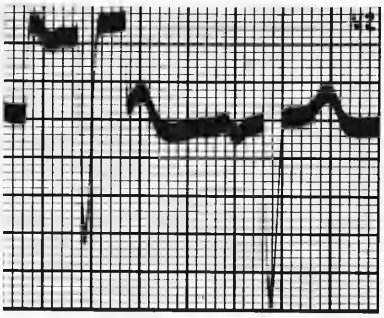


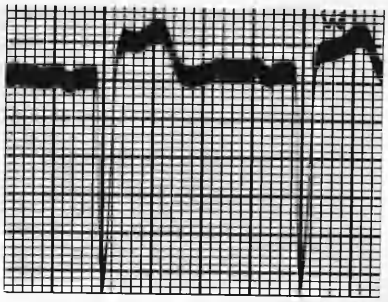


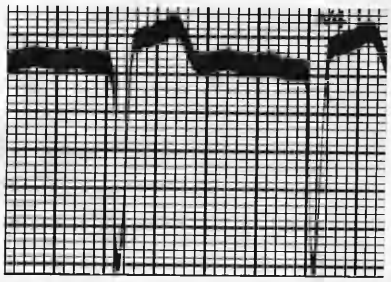


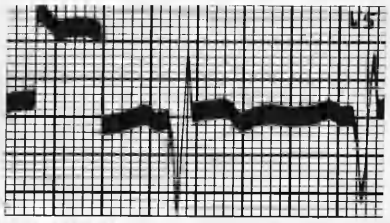
CASE 52, A.R. MALE, 47 YEARS.
15 MONTHS
AFTER ONSET.

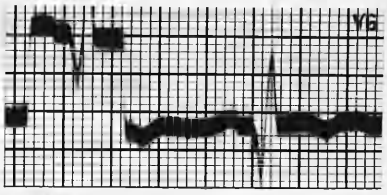


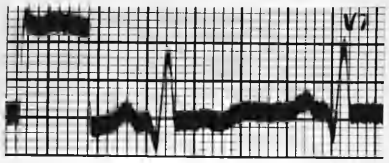












2¼ YEARS
AFTER ONSET.

COMPARISON of STANDARD LIMB LEADS and UNIPOLAR LEADS in the DIAGNOSIS of ANTEROPOSTERIOR INFARCTION

Myers et al (1949) found fifty-two cases of coexistent infarction of the anterior and posterior walls of the left ventricle out of a total of one hundred and sixty-one cases of infarction. They found that the standard limb leads were of very limited value in the diagnosis of coexistent anteroposterior infarction. In only four of the fifty-two cases were the signs in the standard limb leads "strongly suggestive" of both lesions; in thirteen of the fifty-two they were not indicative of either lesion. As a rule the signs of the posterior infarction appear in leads II and III but praecordial leads are necessary for the diagnosis of the anterior lesion.

There are three cases of anteroposterior infarction in the present series of cases. Two of the three are acute cases; in those leads which are affected by the posterior part of the infarction the evolution of the signs runs parallel with that of the /

/the signs attributable to the anterior part, so that it is probable both lesions were of even date and formed parts of one large infarction. According to Wolferth and Wood (1935) the classical pattern of anteroposterior infarction consists of abnormal Q waves in all three standard limb leads, accompanied, early, by elevation of the RS-T segment, greater in lead II than in leads I and III, and, later, by inversion of the T waves throughout. This pattern is presented by case 50; there are classical Q and RS-T signs one day after onset and all three T waves are inverted in the tracing obtained five weeks after onset. However apart from the electrocardiogram obtained one day after onset and which clearly represented a spread of infarction in comparison with that obtained six hours after onset, standard limb lead I does not show a Q wave so that the diagnosis of a localised posterior infarction complicated by generalised pericarditis might have been made.

The other acute case (No. 51) shows an incomplete pattern, insofar as standard limb lead III does not show a Q wave; otherwise it conforms closely to the classical data, viz. a Q (or QS) in lead I, a /

/ a Q wave in lead II, elevation of RS-T in all leads, but the degree of elevation is not greater in lead II than in lead I, probably because the first tracing was not obtained until three weeks after onset by which time the T waves are inverted in all three leads. Thus consideration of the standard limb leads alone would justify the diagnosis of anterior infarction with generalised pericarditis in the tracing at three weeks after onset. Later tracings might be interpreted as anterior infarction alone. In the third case (No.52), the first tracing was not obtained until fifteen months after onset when the pattern of a poorly healed posterior infarction is clearly presented by the standard limb leads. However, the borderline Q wave and low T wave of lead I would raise the possibility of an anterior lesion, or, alternatively, if the heart were in the vertical position, a posterior infarction would account for these signs in lead I. That such was not the position of the heart is seen from the unipolar leads which place the heart in the horizontal position with counterclockwise rotation, as described in the text of the report.

Thus/

/ Thus the three cases demonstrate the variability and lack of specificity of the signs in the standard limb leads in anteroposterior infarction.

S U M M A R Y.

The standard limb leads of three cases of anteroposterior infarction are discussed. In the first the classical electrocardiographic pattern is presented; in the second, the signs are predominently those of anterior infarction and in the third, of posterior infarction.

B.

UNIPOLAR ELECTROCARDIOGRAPHY as an AID to DETERMINING SITE, EXTENT and HEALING of ANTEROPOSTERIOR INFARCTION

As indicated in section A, the diagnosis of anteroposterior infarction requires the employment of both unipolar limb and praecordial leads. While it is true that the standard limb leads may present various signs of infarction, they have little localising value. The posterior part of the infarction requires diagnostic signs in the left leg lead for its detection, whereas the involvement of the anterior wall is attested by diagnostic signs in the praecordial leads.

The left thigh lead of case 50 shows a splintered QS deflection with elevation of the RS-T segment and, later, inversion of the T wave. As explained in the text, the interpretation of these signs in this case is infarction of the posterior ventricular wall. This case presents the anomaly of a temporary reversion to a normal QRS in lead VF, due either to change in electrical position or to spread of infarction in the opposing ventricular wall. It also illustrates the need for caution in pronouncing involvement of the posterior aspect of the apex, if it is

/ is directed backwards, for thus the potentials of the lower border of any anterior infarction might be transmitted to the left leg.

Case 51 presents a typical marginal pattern in lead VF. The cardiac apex is not directed backwards in this case so that the signs in lead VF may be taken to indicate infarction of the posterior portion of the apex. Although the QRS complex barely measures five millimetres, the Q wave is well defined and equals the R wave in size. At the stage of healing, the pattern of lead VF is within normal limits.

In the third case (No. 52) which is of some standing, the signs in lead VF are clearly those of a conduction defect in the posterior ventricular wall.

The anterior part of the infarction is clearly seen in the praecordial leads in all three cases. In lead V1 the small initial R wave is unaffected in all three, indicating intact septal function and any disturbance of the ST-T formation is minimal in this lead compared to that of leads further to the left. The sequence in case 50 is typical of a severe anterolateral lesion with, finally, a fair degree of recovery at both its medial and lateral edges. Leads VR and VL are typical of leads which face the back of the heart in the presence of anterior infarction. In the second acute case (No. 51), the records of which, however, begin at a later date after

/ after onset, the signs of infarction are as striking in leads overlying the lateral ventricular wall as they are in those over the apical region. Hence the left arm lead shows a central pattern. Lead VR with its largely positive ventricular complex is typical of that lead in the presence of a large anterior infarction. The severe involvement of the lateral wall may account, by a reciprocal effect, for the high T waves in lead V2, which in some of the records, shows absence of its R wave due to the anterior infarction. At the stage of recovery, there is considerable recession of abnormal signs in leads V6 and V7 but the usual residual stigma of a previous anterolateral infarction, viz. a deep QS deflection, is seen in leads V2 to V5 inclusive.

Case 52 represents a further stage in the healing of extensive anterolateral infarctions. A deep QS deflection is seen in leads V3 and V4. In the first tracing there are signs of a gross conduction defect in the axillary leads whereas in the second, the development of a late R wave in these leads indicates some degree of subepicardial regeneration. The appearances are closely similar to two cases : No.10 an anteroseptal and No.19 an anterolateral infarction, previously described. Because of counterclockwise rotation and horizontal position, lead VL subtends an unaffected portion of the lateral wall whereas lead VR shows the largely positive splintered ventricular complex, which commonly /

commonly occurs in this lead in the presence of a large anterior infarction.

Thus in all three cases the anterior portion of the lesion is an extensive anterolateral infarction. There are no signs of involvement of the septum. The evolution of the signs is closely similar to that of anterolateral infarction when it is the sole lesion. In one instance the signs of the anterior part of the lesion in lead V2 may have been affected reciprocally by the presence of the severe lateral lesion, not by the co-existence of the posterior extension.

The evolution of the signs in lead VF follows the sequence which occurs when it is the sole lesion.

SUMMARY

Three cases of anteroposterior infarction are analysed in respect of the extent of the lesion and of the evidence of healing as indicated by unipolar limb and praecordial leads. All three shows the signs of an extensive anterolateral infarction in the praecordial leads and of a posterior infarction in lead VF. In one case the signs of posterior infarction in the left leg lead are temporarily suppressed by a spread of the anterior

infarction. Otherwise both portions of the infarct in all three cases evolve in the manner of an anterior or a posterior infarction when present alone.

SECTION VII

STANDARD LIMB LEADS and UNIPOLAR LEADS in LATERAL INFARCTION

The inadequacy of the standard limb leads and of the customary unipolar leads in the diagnosis of infarction confined to the lateral wall of the left ventricle was recognised by Wilson and his school (1946), who selected additional praecordial points as sites for the exploring electrode, viz. at the intersection of vertical lines through the V4, V5, V6 and V7 positions with a horizontal line through the anterior ends of the fourth, and sometimes also of the third, left interspaces. They employed these leads when the left arm leads showed a QR or QS complex, suggestive of infarction, while the customary praecordial leads were non-diagnostic. With the use of leads taken at higher levels they subdivided cases of lateral infarction into "high anterolateral," if the signs were chiefly in the high midclavicular or anterior axillary leads; "high lateral," if they were confined to the high axillary leads and "high posterolateral," if confined to the high posterior axillary leads. The last group was distinguished further by reciprocal exaggeration of the R and T waves in leads V1, V2 and V3.

Myers et al (1949b) found twenty-seven cases with infarction confined to, or localised principally in, the /

/the lateral wall of the left ventricle in his total series of one hundred and sixty-one cases. They found that the diagnostic difficulty presented by lateral infarction varied with its anatomical situation. If it involves the apical one third of the wall, abnormal QS patterns may appear in the customary leads, viz. V5, V6 and/or VL and the localisation of infarct is evident. In the same region, patchy subendocardial or mid-zone infarction may cause no QRS abnormality but there is depression of the RS-T segments of leads V4, V5 and V6.

Difficulty arises in infarction involving the basal one half of the lateral wall. Fourteen of Myers' twenty-seven cases were thus localised. He noted the rarity of abnormal Q waves in leads V5 and V6 and contrasted this finding with their frequency in anterolateral infarction because it generally involves the apical rather than the basal region of the lateral wall. Eleven of the fourteen cases displayed QR patterns in lead VL at least suspicious of lateral infarction and in four of the eleven, high axillary leads were taken. In one, these leads were pathognomonic of infarction; in the other three, they "aided in establishing the diagnosis and in localising the infarct."

Tulloch (1950) has recently analysed the electrocardiograms of thirteen patients with high posterolateral infarction. He finds two distinctive patterns; in the first, diagnostic signs of infarction occur in lead VL and/

/ and standard limb lead I; the R wave is predominant in lead V1; there is no definite transitional zone; there is exaggeration of the T waves in two or more praecordial leads. If however there is concomitant left ventricular hypertrophy, these signs tend to be suppressed . In such a contingency he places some reliance on RS-T depression carried further to the right than in left ventricular hypertrophy alone and on reduction in height of the R wave of lead VL while those of leads V5 and V6 remain tall.

There are two cases (Nos. 53 and 54) of lateral infarction in the present series. The standard limb leads of both have non-specific abnormalities confined to lead I. Both show small inverted T waves and case 53 shows a tiny Q wave. The pattern would raise the possibility of previous anterior infarction and would call for additional leads.

The clinical diagnosis of lateral infarction rests largely on the signs in lead VL. This lead derives its potentials from the lateral aspect of the left ventricle in horizontal, semihorizontal and intermediate hearts: the equiphasic RS pattern in lead VF indicates that in both cases the electrical position falls within this range and hence the abnormal QR pattern displayed by the VL leads may rightly be attributed to infarction of the lateral wall. That the QR pattern is abnormal may be deduced from the following facts. Firstly the Q wave is fifty per cent of the succeeding R wave. This is the ratio required by /

/ by Goldberger (1949) but Myers (1949d) is satisfied if the ratio "exceeds twenty-five per cent". Secondly, the upstroke of the R wave is delayed in both cases - a point stressed by Myers. In case 53, the Q wave clearly occupies almost 0.04 seconds, as required by both standards. In both cases the RS-T changes are inconspicuous, since the infarcts are of some age. The failure of the Q wave of lead VL to cause a Q wave in standard limb lead I in case 54 is due to the immediate negativity of the right arm; the same explanation may be valid for the minuteness of the Q in lead I of case 53. Myers found absence of a Q wave in lead I for the same reason in five out of eleven cases, all of which showed a Q wave in lead VL. However the pattern of infarct in lead VL is the same, whether the lesion is strictly lateral or whether it is the lateral continuation of an anterior or of a posterior infarction. The last-named possibility may be ruled out in both cases under discussion because lead VF is within normal limits. The anterior praecordial leads, by virtue of their retained R waves, provide the evidence that the anterior wall is not involved by the infarction but in both instances the praecordial leads show changes in the T waves due to ischaemia; in case 53 these are confined to the axillary leads and in case 54 they are seen in leads V3 to V6. Reciprocal exaggeration of the R and T waves in leads V1, V2 and V3 is

/ is present in case 53, the pattern of which closely conforms to that described by Tulloch (1952) for high posterolateral infarction. The final pattern in case 54 is less definite because signs of left ventricular dilatation or hypertrophy have developed and, as pointed out by Tulloch, these tend to suppress the evidence of posterolateral infarction. In this case high axillary leads might have been helpful.

SUMMARY

Two out of fifty-nine cases of infarction are lateral in situation. The diagnosis requires an abnormal QR deflection in lead VL. The customary V leads indicate absence of anterior infarction on the one hand and proximity to the ischaemic zone on the other. In one case no further localisation is possible and high axillary leads are indicated. In the other case the pattern conforms closely to that of high posterolateral infarction.

CASE 53. J. McC., 39 years, male.

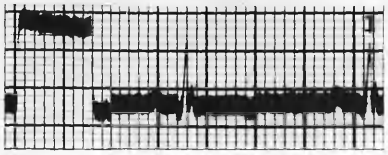
This patient first attended the Out-Patient Department six and a half weeks after onset of illness. He had had sudden praecordial pain radiating down the left arm and accompanied by vomiting; it had lasted two days. When examined at the Out-Patient Department blood pressure was 110/85 and the cardiac sounds were of indifferent quality.

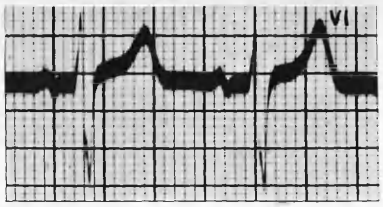
An electrocardiogram shows left axial deviation with very slight upward convexity of the ST segment in lead 1 and inversion of the T wave which is however very small. The Q wave is inconspicuous. Thus the findings in lead 1 are no more than suggestive of recent anterior infarction. The unipolar limb leads indicate that the infarction involves the lateral wall since in lead VL there is a broad Q wave measuring half of the amplitude of the succeeding R wave which is delayed and slurred; there is slight shouldering of the ST segment and the T wave is small and inverted. Since lead VR shows the configuration normally derived from the back of the heart, the possibility that the pattern of lead VF may have a similar source must be considered especially in view of the inversion of its P wave which is typical of posterior leads. However /

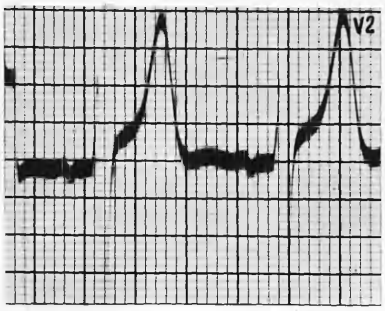
/ However the duration of the Q wave from onset to nadir is as long as 0.04 sec. and the slurring of the R wave is so conspicuous that, irrespective of its basic pattern, these signs are almost certainly due to subendocardial infarction. Lead VF is normal for an intermediate or semihorizontal heart. The anterior praecordial leads show the unduly prominent R waves and tall spike-like T waves which are typical of these leads when they face an infarction in the opposite wall. Hence either the lateral infarction has extended into the posterobasal region of the left ventricle or there has been severe clockwise rotation so that the anterior praecordial leads are in apposition to the lateral infarction itself. The fact that lead VR faces the back of the heart and the possibility that lead VL may also be in a similar relationship favour clockwise rotation, as does also the displacement of the transition zone in the praecordial leads to the left. Lead VF gives no indication of any infarction in the posterior wall but such a negative finding is not infrequent when the basal part of the posterior wall is involved. Similarly /

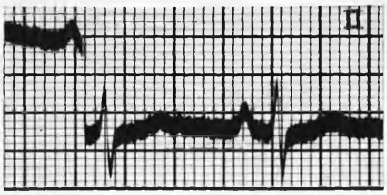
/ Similarly the paucity of information to be gained from leads V5 and V6 is typical of lateral infarction since it frequently spares the apical one third of the anterolateral wall. The diphasic T in lead V5 and the inverted T in lead V6 are indicative of the zone of ischaemia surrounding the lateral or the posterolateral part of the infarction.

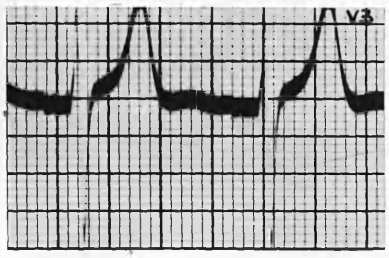
This case is therefore an example of a high lateral infarction with possible posterior extension.

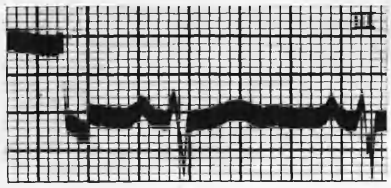


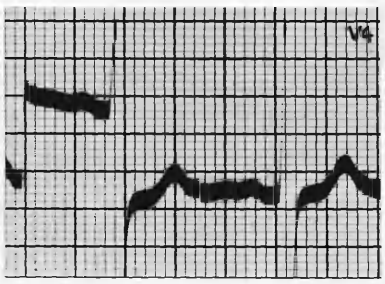


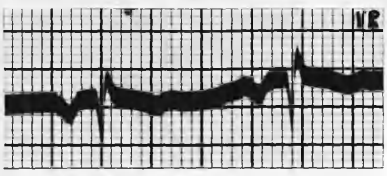


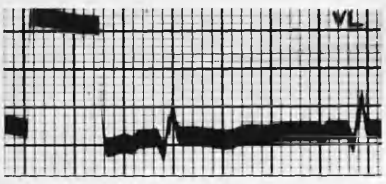


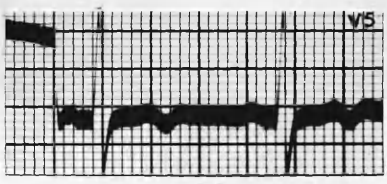


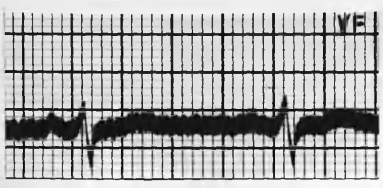


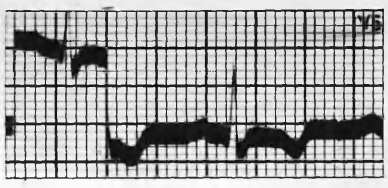












CASE 53, J.McC., MALE 39 YEARS.

6½ WEEKS AFTER ONSET.

Case 54, J.A.L., male, 50 years.

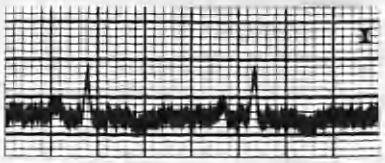
This patient, a clergyman, had had indifferent health for the six months previous to the first electrocardiogram. He had had recurrent anginoid pain and dyspnoea following an acute illness, which had been diagnosed as coronary thrombosis. When seen as an outpatient, there was slight facial cyanosis. Blood pressure was 160 systolic, 100 diastolic. Cardiac enlargement was not demonstrable clinically. The cardiac sounds were of average quality.

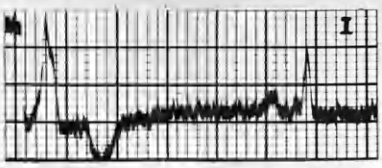
The first electrocardiogram shows sharp inversion of the small T wave in lead I; there is no Q wave because of greater initial negativity of the right arm. Lead VL shows a QR pattern, the Q wave being half of the size of the following R wave, thus fulfilling the criteria of Goldberger (1949) for QR complexes in lead VL attributable to infarction. Furthermore, the RS-T segment is bowed upwards and T is inverted. These signs comprise the fixed pattern of infarction in the underlying lateral wall of left ventricle. The praecordial leads show preservation of the R wave in leads V2, V3 and V5, thus indicating that the anterior cardiac wall is not involved by the central zone of infarction. On the other hand, signs derived from the ischaemic zone are clearly seen in the praecordial leads, viz. inverted or flat T waves in leads V3 to V6 inclusive. There is no Q wave in the/

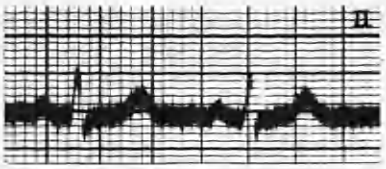
/in the axillary leads.

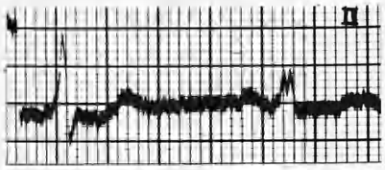
The patient was not seen again until one and a half years later. Health was degenerating. Dyspnoea occurred readily and angina was frequent. The cardiac sounds were indifferent and radiologically there was "slight prominence of the left ventricular shadow but the transverse diameter of the heart appears just within the limits of normal."

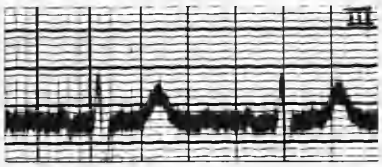
An electrocardiogram now shows frequent ventricular extrasystoles. The left arm lead still shows a blunt Q wave but the R wave is much taller so that the ratio of Q/R no longer fulfils the requirements of infarction. The increase in the R wave may be explained by the signs of left ventricular dilatation seen in the praecordial leads - the very deep S waves in the anterior leads and the prominent R waves with shelving RS-T segments in the axillary leads. The latter sign may probably denote local subendocardial ischaemia. Were it not for the preservation of the R wave anteriorly, the general pattern is that of a poorly healed anterior infarction.

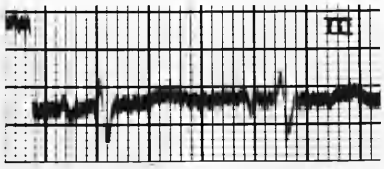


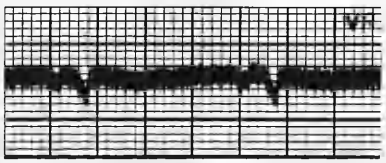


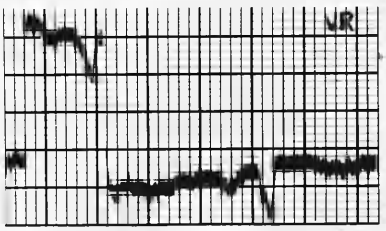


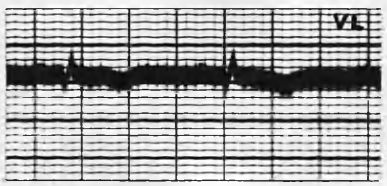


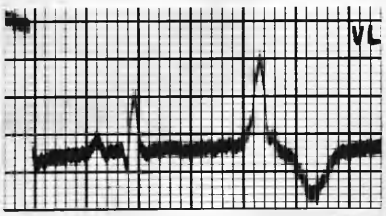


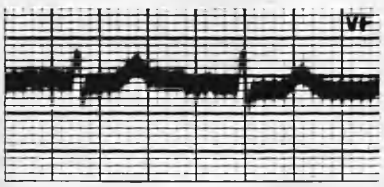


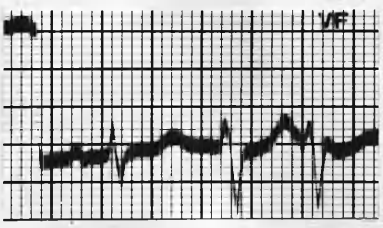








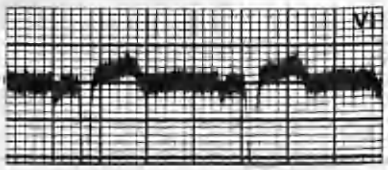


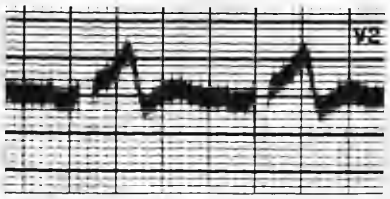


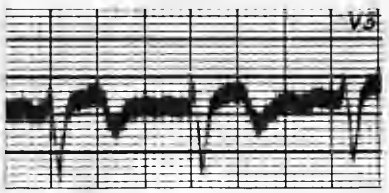
CASE 54, J.A.L. MALE, 50 YEARS.

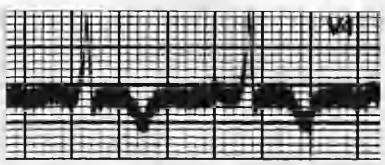
6 MONTHS
AFTER ONSET.

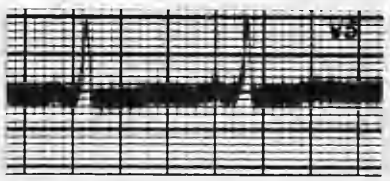
2 YEARS
AFTER ONSET.

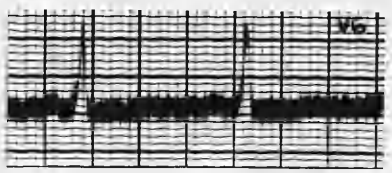








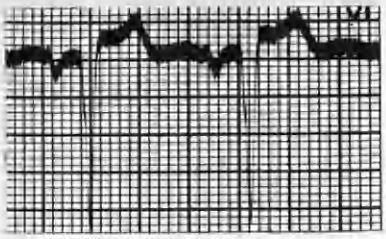


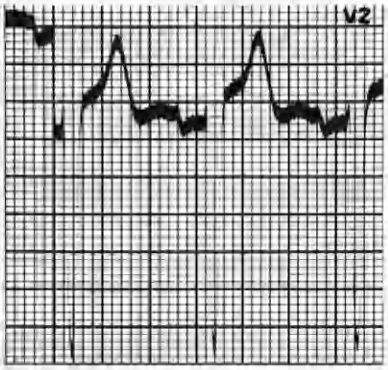


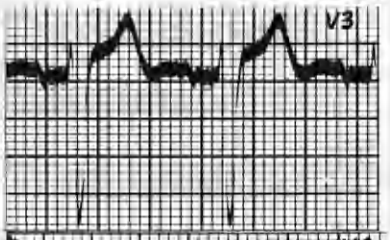
CASE. 54, J. A. L. MALE, 50 YEARS.

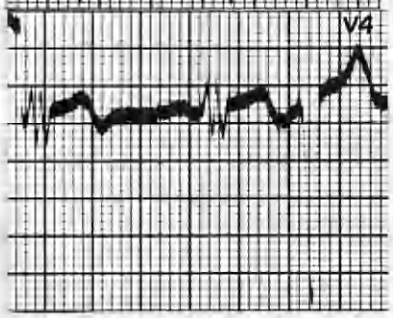
6 MONTHS

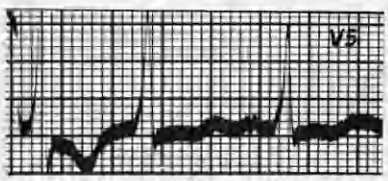
AFTER ONSET.

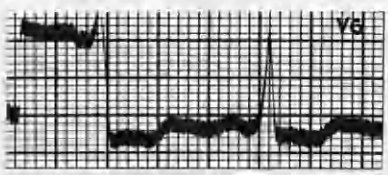


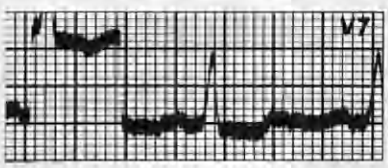












2 YEARS

AFTER ONSET.

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