The remarkable clinical improvement of patients with breast cancer which sometimes follows hypophysectomy or adrenalectomy and oophorectomy is still not satisfactorily explained.

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AN ANALYSIS OF 161 CASES OF CARDIAC INFARCTION WITH ABC ELECTROCARDIOGRAPHIC STUDIES AND 36 NECROPSIES*

BY

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In the *Lancet* (1954), in a section concerning vectorcardiography and referring to the ABC display, it was stated that "satisfactory electrocardiograms have been obtained from cases of known heart disease, but confirmation from larger numbers is needed." In the present paper 161 cases of cardiac infarction observed over a period of 2-8 years by the ABC display have been studied with this end in view. Necropsies were performed on 36 cases of this series.

The standard unipolar limb and chest leads record electrical activity almost solely in the frontal (coronal) plane. The vectorcardiograph is limited with regard to the study of temporal relationships and the acceptance of a standard frame. This simple three-dimensional display, allowing a three-dimensional analysis, is not deficient in these respects. As will be seen from this survey, it detects all changes detected by the present normal leads, and in addition detects changes not demonstrated by the normal leads.

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The series of patients is classified into the following groups.

A. (i) Clinically diagnostic, standard E.C.G. diagnostic, ABC leads diagnostic: 127 cases; 32 traced deaths, 25 necropsies. (ii) Clinically diagnostic, standard E.C.G. not diagnostic, ABC leads diagnostic: 24 cases; 5 traced deaths, 5 necropsies.

B. Clinically not obvious, standard E.C.G. not diagnostic, ABC leads diagnostic: (i) fatal cases 4, 2 necropsies. (ii) survivors 2.

C. Infarct totally unexpected, ABC leads diagnostic: 1 case (necropsy positive).

D. Clinical diagnosis infarction, ABC leads negative: 1 case (necropsy negative).

E. Special instances: (i) Minor infarct patterns: 4 cases; 1 traced death, 1 necropsy. (ii) Abnormal T-wave form (also included in A): 1 case. (iii) heart block (also included in A): 17 cases; 12 traced deaths, 9 necropsies.

Method

The majority of the patients were examined personally by me, and the condition of all patients was discussed with at least one physician concerned with the management of each patient.

FIG. 1.—(a) Planar sheet (diagrammatic) of infarction AB at right-angles to YOY. Further details in text. (b) Linear infarction, C, at right-angles to A and B axis. Further details in text. (c) Four dipoles paired and orientated at 180 degrees. Further details in text. (d) Clockwise dipoles. Further details in text. (e) Direction cosines (0,0,1; 1,0,0; 0,1,0) to ABC axis. Further details in text.

E.C.G. recording was usually performed with a Both direct writing electrocardiograph and in some instances with a "cardio-scribe" direct writing machine. A continuous check was made to ensure that the recording apparatus was kept in good condition.

When patients died every endeavour was made to have a necropsy performed, though this was not always possible owing to conditions beyond our control. In some instances the freshly removed intact heart was perfused through the coronary vessels with a gelatin-barium suspension at 37° C. and 120 mm. Hg pressure and then x-rayed as described by Blumgart *et al.* (1940).

ABC electrocardiographic recording was made from the right-angled system: xiphisternum (X), manubrium

sterni (M), left axilla (L), and right chest at the base (R), as described previously (Trethowie, 1953). Lead A is taken from the manubrium and xiphisternum (MX), lead B from the left mid-axilla at the level of the xiphisternum and the xiphisternum (LX), lead C from the right chest at the base at the level of the xiphisternum and the xiphisternum (RX). LX, MX, and RX are all mutually at right angles. The electrode connected to X is positive for upward deflection and thus corresponds to the left leg lead in lead II Einthoven. Thus the right arm lead corresponds to the varying electrode placed successively on the manubrium, left axilla, and right chest at the base. This should define electrical fields in a three-dimensional conductor when these fields emanate from a dipole within the volume, and probably also would analyse whatever combinations of dipoles are present or whatever the characteristic electrical events arising in the conductor. Certain modifications occur owing to the fact that the medium is not homogeneous, though nearly so, and the conductor is not infinite, but bounded by the body surface. The former modification is regarded as minor by most observers. Schmitt (1955) has shown there is a modification, and actually recorded it, with a homogeneous medium containing a dipole inside a model in the shape of the human torso.

Influence of Infarction on the E.C.G.

Let us consider a planar sheet of heart muscle which is infarcted (Fig. 1,a). Such a sheet produces a "current of injury," the injured portion of muscle being at the surface electrically negative compared with the surface of the adjacent healthy muscle. The QRST complex then shows elevation of the RT segment, provided the leads chosen are in the plane of the affected muscle (AB). In the line YOY, at right angles to this plane for all practical purposes, the heart contains only healthy muscle, and segment elevation will not be observed in a lead chosen along this direction—for example, lead C (Fig. 1,a). Depending on the angle formed by the planar sheet to the directions A, B, and C, so we will obtain more or less "weighting" or segment elevation in specific leads.

Let us next consider a linear distribution of infarction (Fig. 1,b). In this instance no effects will be detected in leads A, B, but considerable change will be detected in lead C. If C were only slightly askew to the plane AB there would be relatively little observable effects of the infarction in C. This is the case with V_{1-3} with small septal infarcts. Such infarcts do, however, show gross changes in lead C of the ABC display.

Since the posterior aspect of the heart forms a considerable vertical sheet of muscle, infarcts in this area classically produce maximal effects in lead A. The antero-lateral aspect of the left ventricle which recedes posteriorly and cranially is parallel to lead B, and thus infarcts

in this region produce maximal changes in that lead. The septum is almost parallel to lead C, and infarcts in this portion in the heart produce maximal effects in lead C. Likewise the AC pattern indicates posterior septal infarction and the BC pattern indicates anterior septal infarction. This aspect is amplified later with necropsy studies (cf. Fig. 2).

To produce a null effect as referred to above, dipoles would be required to be orientated in opposite directions (Fig. 1,c). However, cardiac infarction simply produces a steady state of "injury" current which gradually subsides over two to three weeks with the development of fibrosis followed by contraction. This latter phenomenon alters the axis of adjacent healthy muscle fibres and may result in a permanent inversion of the T wave in a particular lead. Ischaemia may also affect the T wave. Two areas of infarction would summate this "negativity," not cancel each other out. Similarly a null effect could be produced by a ring of muscle with a clockwise (or anti-clockwise) orientation of dipoles, and dipole α and dipole β (Fig. 1,d) might then cancel out one another; but again the effect of infarction, as before, is to produce

FIG. 2.—Infarct sites, as detected at necropsy. Continuous line, recent infarction; broken line, discrete fibrosis; dotted line, degeneration only. Top left-hand corner, Patient number, E.C.G. infarct pattern designated centrally.

FIG. 3.—(a) Patient 93: Septal infarction extending anteriorly over the left ventricle with fibrotic lamina from older infarction parallel to the right. Further details in text. (b) Roentgenogram of perfused heart from Patient 128. Right coronary block at A and left coronary at B. Further details in text.

a steady "state" of negativity. Contraction of infarcted muscle does not occur with heart beats. In the presumed circus movement of flutter, now not acceptable in its original form, the repetitive firing of the P wave is the diagnostic feature, not injured muscle. Further, the nearer bipolar leads are to the heart the less likely is it that clockwise dipoles would cancel one another out. The ABC leads are close to the heart.

For changing potentials—that is, when currents are produced as distinct from static charge—the field patterns will not be arrived at immediately because there is a degree of impedance in the conductor (that is, the body). However, Kaufman and Johnston (1943) consider these variations in resistance as minor, and that they do not influence potentials greatly. Further preliminary tests on a human torso with a dipole source at the heart position show that the potentials as recorded for leads A, B, and C, while not being at the precise level expected, are nevertheless at 60%, 75%, and 90% of the theoretical value, and, so long as an abnormal contour appears even in part, this should be large enough to have practical significance. Again the particular problem concerning us here is the evaluation of infarcts, and these produce a steady current of injury, so that estimates based on static laws probably do apply here. The problem is best answered by the practical results obtained: namely, do the leads used record significant changes in accord with pathological findings?

It would appear axiomatic that a right-angled distribution of the ABC type is the logical display to choose for electrocardiography because no electrical axis is then unrepresented and more obvious segment elevation must be produced in one lead than is the case with any other display owing to that distribution. In the representation shown in Fig. 1,e, the direction cosines become 0,0,1; 0,1,0; and 1,0,0, when the ABC leads correspond to the axes chosen in the original figure, because $\cos 90^\circ = 0$ and $\cos 0^\circ = 1$. The approximate size of the infarct can then be indicated. In point of fact point X (xiphisternum) is close to 0 (Fig. 1,e), since the heart occupies the anterior part of the mediastinum, and this distribution applies as nearly as is possible practically. Further, this distribution, being located on skeletal points—namely, manubrium and xiphisternum—ensures regularity of repetition of the display from patient to patient.

E.C.G. Pattern and Infarct Site as Revealed at Necropsy

The sites of the infarct in 36 instances where necropsy studies were available are shown in Fig. 2. In these the heart has been cut across horizontally below the level of the atria and observed from above, so that the left ventricular cavity appears on the left and the right cavity on the right. The photographic findings of the heart from Patient 93 are shown in Fig. 3,a, where two parallel septal infarcts of different age are well shown. Leads ABC showed a "Cb" type cardiac infarction, indicating recent septal involvement extending on to the antero-lateral surface. Also R_A was absent and without segment elevation in A—that is, Q_A form—indicating older infarction in the posterior portion, and in fact there was older septal infarction extending posteriorly to involve both ventricles at a lower level than that shown in Fig. 3,a. It will be observed that, as expected from theory (Trethewie, 1953), "C" type infarcts are largely septal (Fig. 2), "B" type infarcts more commonly involve the antero-lateral aspect (Fig. 2), and "A" type infarcts more commonly involve the posterior aspect of the heart (Fig. 2). Occasionally BC type infarcts involve the posterior and medial aspect of the left ventricle rather than being antero-septal, especially if block is present. Thick black lines indicate recent cardiac infarction, interrupted lines old infarction (with fibrosis), and dotted lines degeneration only (Fig. 2).

X-ray Studies: Radio-opaque Perfusion

In five instances the heart was perfused with radio-opaque material, laid open by incision along the anterior border between the two ventricles, the septum excised, and the whole x-rayed. Fig. 3b was obtained from Patient 128, and a block is shown in the right coronary artery (A) after giving off the inferior interventricular branch and also in the anterior descending branch of the left coronary artery (B). The E.C.G. (Fig. 4) indicated old posterior infarction (Q_A) and recent antero-lateral infarction involving the septum (ST_{BC}). At necropsy there was an old posterior infarct, and in the postero-medial aspect of the left ventricle (including the septum) there was recent infarction—that is, parallel, but posterior to the region suggested.

FIG. 4.—E.C.G. of Patient 128, heart depicted in Fig. 3: "Bc" type infarct pattern with old Q_A .

Classified Groups

A. Cardiac Infarction Obvious Clinically

(i) *Patients with histories indicative of cardiac infarction associated with typical electrocardiographic tracings.*—This aspect need only be discussed quite briefly, and it is sufficient to point out that in 127 patients there was no doubt whatever that the E.C.G. standard display (E₁, E₂, E₃; aVR, aVL, aVF; V₁₋₆) yielded conclusive evidence of infarction. Leads ABC also yielded conclusive evidence of infarction in all 127. One impressive feature has been the magnitude of the segment elevation in the ABC display, which is frequently greater than that of the standard display, and the fact that changes are so commonly seen in a greater proportion of leads with the former. Definite pattern

FIG. 5.—E.C.G. of Patient 26: "CBa" type infarct pattern.

changes are observed in at least one lead in 100% of cases, in at least two leads in 86% of cases, and in all three ABC leads in 39% of cases. One of the latter is shown in Fig. 5, which is the tracing obtained from Patient 26, who developed sudden substernal pain at rest five days before admission with a very severe recurrence four hours before admission on May 18, 1955. This tracing is recorded as a "CBa" type infarct pattern (Trethowie, 1953)—that is, there are gross changes in leads C and B, though greater in C, and definite but smaller changes in lead A. The infarct is septal and extends on to the antero-lateral surface. This patient developed a second infarction two months later ("Abc" type)—that is, posterior infarction—and he died on the fifth day of the attack. Necropsy was not permitted.

(ii) *Patients with histories indicative of cardiac infarction associated with standard display tracings not regarded as indicating evidence of infarction, ABC tracings diagnostic.*—Twenty-four patients (15% of cases) came under this heading. In each case leads ABC yielded unequivocal evidence of infarction. Such an instance is recorded with Patient 106. Three days before admission this patient, a male, complained of the sudden onset of severe pain under the sternum, with collapse. He was sweating, and his colour for a time was somewhat greyish. Twelve days after the onset his B.S.R. was 118 mm. in one hour and 128 mm. in two hours. His standard E.C.G. eight days after the onset is recorded in Fig. 6. The ST segment in V₁₋₃ is not quite normal, but this was not regarded as pathognomonic of infarction. The T-wave in V₅ and V₆ is of low potential and inverted. Leads ABC (Fig. 6), taken at that time, show conclusive evidence of infarction of the Bac type with maximum segment elevation in lead B—that is, antero-lateral cardiac infarction.

The tracing shown in Fig. 7 was obtained from Patient 80, who had attacks of substernal pain on the two days before and on the day of admission to hospital. Four hours before admission the pain was severe, prolonged, and associated with collapse. The standard E.C.G. throughout the course of his illness was recorded as not signifying infarction, but a confident diagnosis of infarction was made on clinical grounds. On the nineteenth day the tracing of leads ABC

FIG. 6.—ABC-E.C.G. and standard E.C.G. of Patient 106. Tracing taken eight days after onset. Note the considerable segment elevation in B not apparent in standard leads.

was taken (Fig. 7). This shows segment elevation in all leads, maximal in lead B with obvious elevation in lead C with "round-shouldering." Elevation is also present in minor form in lead A. This is the distinctive feature confirming the diagnosis of cardiac infarction in contradistinction to left-heart-strain pattern. In this instance, of course, a confident clinical diagnosis was possible, but the value of electrocardiography, especially if it can be shown to be correct regularly in the absence of a clinical history suggesting infarction, is self-evident, and is discussed in the next section. A similar finding is shown in Fig. 8 (Patient 20), where the tracing was obtained 30 hours after the onset of infarction and segment elevation is considerable, while the standard leads E.C.G. did not indicate infarction.

The infarct pattern in the 24 cases coming under the above heading showed the distribution recorded in Table I.

For clarity, a "BC" infarct pattern—that is, marked change in B and C, though greater in B—includes CB (changes greater in C); likewise with BA and CA, and in 12 instances where some change was observed in the third lead also, the notation was recorded as the two leads in which the changes were greater. It is noteworthy that in only one instance in the 161 patients did lead A (indicative of posterior infarction) yield conclusive evidence of infarction when the standard leads did not. In this case block was present which obscured the diagnosis in the standard leads. This

FIG. 7.—E.C.G. of Patient 80, showing typical infarct pattern on the nineteenth day; standard lead tracing not indicative of infarction throughout course of illness.

TABLE I

Infarct type	BC	BA	B	CA	A
No. of tracings	12	5	4	2	1

aspect will be discussed later. On the contrary, in 14 instances (BC and CA type pattern) where the septum or that portion of the left ventricle parallel to it was involved, conclusive evidence of infarction was manifest in leads ABC, while the standard leads did not yield conclusive evidence of infarction. Of ten patients who died and on whom a

FIG. 8.—E.C.G. of Patient 20; typical infarction 30 hours after onset. Standard leads did not indicate infarction.

expected. Standard lead tracings, where minor changes were observed, which were not regarded as presenting evidence of infarction but were nevertheless not normal, involved leads V_1 , V_2 , or V_3 in four instances, lead 3 in one, and lead V_4 in one. In the remainder changes were quite trivial.

Changes found in lead V_3 of the type referred to above are probably due to the infarct, but since this lead is only slightly askew to the frontal plane, as referred to earlier, infarct pattern changes are not likely to be marked if the frontal plane leads (Einthoven and lateral V leads) show indefinite infarct patterns.

Five of these 24 patients died. Necropsy was performed on all five subjects, and the infarct was found in the site expected for the ABC finding in each instance.

In 5 of these 24 instances (3% of cases) the initial diagnosis made was of cardiac infarction, but considered opinion at a later date was opposed to this diagnosis. In each instance, in the opinion of at least one other physician and in my own, these patients were still regarded as suffering from myocardial infarction. Such a state of affairs is to be expected, especially as it is recognized that some 15% of patients do not show conclusive evidence of infarction in standard lead electrocardiography. Emphasis in such cases must therefore be placed largely on clinical findings.

It is pointed out below that one patient who was not regarded as suffering from myocardial infarction on clinical grounds, and who was not considered to have a myocardial infarct after necropsy (macroscopic) examination, was recorded as clearly associated with myocardial infarct pattern in the lead ABC findings. Subsequent histological examination of the heart, carried out because of the latter findings, revealed the presence of a laminar infarct in the posterior part of the heart. Again in Section D, below, an infarct was diagnosed clinically without support in the E.C.G. findings with leads ABC; and subsequent necropsy, including microscopical examination, did not reveal the

necropsy was performed where the pattern was of this type—that is, with major involvement in lead C—the septum was found to be involved in seven, and in three instances the postero-lateral part of the left ventricle (parallel to the septum) was involved. Nine other patients were found to have an associated BA or B type infarct pattern (Table I). Since lead B is inclined at about 40 degrees to the frontal plane, this finding could be

presence of infarction four days later nor was coronary atheroma present. There was also adequate evidence at necropsy for another cause of death. Such findings naturally strengthen one's confidence in the value of leads ABC, and the absence of standard lead confirmation may have played a part in the different clinical opinion expressed in relation to the five patients referred to here. Nevertheless, at the initial examination the clinical picture was regarded by all as being that of cardiac infarction.

The case history of one of these patients is presented as an example.

Patient 53 complained of the sudden onset of tearing pain at the lower end of the sternum and in the epigastrium one week previously. The pain radiated up to the back of the neck. On admission his B.P. was 180/90 mm. Hg. The standard lead E.C.G. was regarded as normal throughout his stay in hospital. Lead 3, obtained on the eighth day after the onset of the pain, showed a minor anomaly considered to be sufficiently like a normal RSR'. The B.S.R. at this time was 35 mm. in one hour and 64 mm. in two hours (Westergren). X-ray examination of the lungs was normal. Leads ABC revealed a classical Pardee curve in leads A and C, indicating a postero-septal infarct.

B. Infarction not Obvious Clinically

(i) *Patients who died where leads ABC showed unequivocal infarction but in whom infarction was not certainly diagnosed on clinical grounds, and the standard leads did not yield definite evidence of infarction.*—Four patients in whom leads ABC showed unequivocal infarction died, while infarction was not certainly diagnosed on other grounds. In each of these patients standard lead E.C.G.s did not confirm infarction. One

of the patients had complained for years of abdominal pain on exertion, and, following a more severe attack of pain, dyspnoea at rest appeared. Her B.P. was then 160/120. From a consideration of the clinical picture and the standard E.C.G. leads the consensus of opinion was that the patient suffered from ischaemia. Slight segment elevation was shown in V_4 , and this was thought not to be indicative of infarction. In leads

ABC, however, a classical Bac type infarct pattern was observed—that is, an antero-lateral infarct (Fig. 9). At necropsy an antero-lateral laminar infarct was found in the left ventricle. This infarct encroached neither on the endocardial nor on the pericardial surface. A histological section is shown in Fig. 10, which shows the infarct clearly.

The second of these patients developed cardiac failure fairly suddenly and was critically ill; it was thought that cardiac infarction could not be excluded. The standard E.C.G. (1, 2, 3, aVL, aVF, V_2 , V_4 , V_6) did not suggest cardiac infarction, but lead C showed gross segment elevation and also a W-shaped contour, including a well-marked Q wave. At necropsy a recent and an old infarct were found in the septum as expected (lead C lies in the septal axis).

Necropsies were not performed on the other two subjects.

(ii) *Patients who survived where leads ABC showed unequivocal infarction, but in whom infarction was not certainly diagnosed before electrocardiography and the standard leads did not yield definite evidence of infarction.*—There were two patients in this group. One patient, a

FIG. 9.—Typical "Bac" type infarct. Clinical history thought to indicate ischaemia only. Standard leads, ischaemia; necropsy, antero-lateral infarct (left ventricle).

woman aged 51, had complained for 15 years of sharp pain near the lower sternum in the evening on walking. She did not stop walking on experiencing the pain. This pain was regarded as not indicative of organic cardiac disease. A standard lead E.C.G. and leads ABC at that time were normal. Her B.P. was 148/80 mm. Hg. Following examination I expressed the opinion that the patient might be suffering from cholelithiasis and advised cholecystography, but that I was not certain whether the patient suffered from angina of effort in addition. Cholecystography revealed the presence of numerous biliary calculi.

FIG. 10.—Microscopical section of heart from patient whose E.C.G. is recorded in Fig. 9. ($\times 543$.)

Cholecystectomy was performed. Two years later the patient stated that the pain had become worse. Then six months later, at a second examination, she said that one week previously she experienced a sudden onset of lower sternal pain and sweated, the pain lasting four hours. A standard lead E.C.G. was normal, but lead A in the ABC display revealed the presence of a classical Pardee curve, indicating posterior cardiac infarction.

C. Cardiac Infarction quite Unsuspected on Clinical Grounds but Evident from Leads ABC

This patient was admitted to hospital with hypertension and developed a hemiplegia. He did not complain of cardiac or chest pain. A standard E.C.G. was not done. Leads ABC (Fig. 11a) show segment elevation with round-shouldering in lead A, conclusive evidence of posterior infarction. There is also segment elevation in leads B and C, but in B the elevation is angulated very much as it is in hypertensive strain. With such a degree of elevation in leads B and C in hypertension ordinarily the S waves are

much larger. Here they are normal to reduced in potential. The T waves are unduly large and abnormal. The patient was 38 years of age. At necropsy the heart, other than showing considerable hypertrophy, appeared normal macroscopically. However, more careful examination of the posterior part of the heart, carried out because of the ABC-E.C.G. finding, revealed the presence of a recent laminar infarct. The patient was also found to have coarctation of the aorta, and for interest a tracing obtained from another patient who had coarctation of the aorta (without infarction) is shown in Fig. 11b. Interestingly enough the S waves again are not prominent and there is flutter-fibrillation (lead A). Segment elevation with angulation is seen in leads B and C, but, more significantly, the S-T segment is depressed in lead A. This is the distinctive feature of left-heart-strain pattern. With cardiac infarction the S-T segment in lead A is not depressed or concave upwards at the isoelectric line. Infarct patterns with leads ABC always show elevation, not depression, of the S-T segment when this is abnormal. Further, the round-shouldering of the contour is significant of infarction. In the absence of segment elevation, as with other leads, infarction is of course indicated by a cove-shaped usually inverted T wave and a biphasic R-T interval. Segment elevation pattern may conform to the monophasic type or the high take-off of the Pardee type.

D. Cardiac Infarction Diagnosed Clinically Without Changes in Leads ABC Suggestive of Infarction

This patient complained of shortness of breath and cough for some months and chest pains one week before admission. Retrosternal pain with cyanosis developed on the previous day, and the patient was treated with anticoagulant therapy for cardiac infarction and also treated for cardiac failure, pneumonia, and shock. The prothrombin level fell to 12%.

The standard E.C.G. was recorded as indicating ischaemia only. Leads ABC did not support the clinical diagnosis of cardiac infarction. The ABC-E.C.G. is presented in Fig. 12. Potentials are very low, as is seen sometimes in pericarditis or in approaching death; the T waves are flat. This patient died 72 hours later. The recording made against the ABC leads before death was: "Very low potential, ischaemia apparent in A; I would think the muscle would show degenerative changes microscopically, but the tracing is not of the Pardee type." It was concluded there was no infarction. At necropsy three small foci of old pericarditis were found. The coronary vessels were free of atheroma, as was the proximal aorta. The ventricular muscle was dark brown in colour, but no infarct was found. Microscopical examination at a later date revealed the presence of brown degeneration in the cardiac muscle. Other significant findings at necropsy were gastro-intestinal haemorrhage (gross), pulmonary emphysema, acute bronchiolitis, cholelithiasis, and diverticulitis coli. The gall-bladder was distended with bile and there was impaction of small pigment calculi. If one

FIG. 11.—(a) Typical posterior infarct pattern in patient with coarctation. (b) Typical tracing of coarctation of the aorta without infarction.

FIG. 12.—E.C.G. of patient with old pericarditis and ischaemia. Further details in text.

can apply an electrocardiographic technique which is so complete that infarction can be excluded if the E.C.G. is negative, obviously one has a most valuable tool to aid diagnosis.

E. Special Instances

(i) *Minor E.C.G. "infarct" patterns.*—It is regarded as likely that, since leads ABC record three leads mutually at right angles with a common origin, the grossness of the infarct can be deduced approximately from the degree of segment elevation all three leads. On the contrary, if only the frontal and slightly skew frontal planes are recorded, gross infarcts may sometimes produce little segment elevation in the electrocardiogram.

A tracing may be observed, in the case of a patient considered to be suffering from cardiac infarction on clinical grounds, where the E.C.G. tracing forms a very small though definite "Pardee" complex or other slight segment elevation. If this tracing is magnified, the evidence of infarction electrocardiographically becomes more obvious. It is suggested such patients developing pain at rest show recent degenerative cardiac changes short of infarction. This feature was observed in four patients in the present series, and is seen more often in private practice. For accuracy of recording, an ink writer forming a fine line, or a cathode-ray oscillograph, is required to demonstrate this feature. Multiple T inversions approaching a cove shape also occur in severe acute ischaemia. All of these patients were diagnosed clinically as suffering from cardiac infarction without any doubt. Three survived. The other patient died. The ABC findings in one of the former showed a Pardee curve in lead B, passing to a cove-shaped inverted T wave and a Pardee curve in lead C. The standard E.C.G. appeared normal, showing an RSR' of apparently normal type in lead V₂. In the remaining two, minor monophasic segment elevation was evident in leads ABC. The ABC findings in the fourth patient showed a curve of Pardee type in lead C, leading to an inverted abnormally shaped T wave. This was less evident in lead B, while there was no segment elevation in lead A (Fig. 13). Magnification of the tracing demonstrated the feature more readily. Similar though less obvious findings were observed in V₂ and V₃. This patient died seven days after admission on August 22, 1955. The ABC-E.C.G. diagnosis before death was "Pardee type tracing: septal infarct, very small." At necropsy broad zones of pallor were observed in the left side of the interventricular septum and in the papillary muscles of the anterior wall. Both coronary arteries were almost completely occluded at their origins, and elsewhere were grossly atheromatous. Microscopically, cloudy swelling and some vacuolation were seen in the muscle fibres in the pale zone, but no massive infarction was observed (Fig. 14).

It is considered worth while to follow such cases through with necropsy findings wherever possible, so as to determine if such minor changes as illustrated in these four examples are indicative of degenerative changes in the muscle as distinct from discrete infarct formation.

FIG. 13.—Minor infarct pattern of Pardee type. Myocardial degeneration in pyriform areas in septum.

Continued reduced blood supply and oxygen lack can be expected to produce degenerative changes. This has been shown for the liver (Trethewie and Gaffney, 1951). Undoubtedly post-mortem changes

require exclusion, and the interval after death before necropsy examination needs to be kept at a minimum even though the body is placed in the refrigerator. In view of the facts that the second subject died during cold weather (in Melbourne), was placed in the refrigerator on arrival at the necropsy department, that bands of pallor were seen in the heart in the area suggested to be involved previously by the ABC findings, that the

FIG. 14.—Microscopical examination of heart from patient whose E.C.G. is shown in Fig. 13. (×554.)

coronary arteries were almost completely occluded at their origins, and that actual vacuole formation was observed in cells in pyriform areas of degeneration, it is considered very likely that the microscopical changes observed in the heart were of ante-mortem origin.

The incidence of this condition in patients admitted to public hospitals with a diagnosis of cardiac infarction is lower than that found in the general population. For instance, apart from the series of 161 cases reported here, of 139 cases of heart attacks seen privately and thought clinically to be possibly or probably due to cardiac infarction, 115 showed frank evidence of cardiac infarction electrocardiographically with leads ABC, and 24 showed these minor changes of "ischaemia at rest." This feature, recognized at the necropsy referred to above performed in August, 1955, has been re-emphasized recently for the standard leads in relation to S-T depression. The polarity of the ABC leads, since point X is close to zero potential (Nahum, Mauro, Chernoff, and Sikand, 1951), probably accounts for the fact that the current of injury in the degenerated muscle, as with frank infarction, also produces segment elevation in the tracing. Frey (1938) and Oettel (1941) describe elevation of RST during acute coronary insufficiency without infarction at necropsy.

(ii) *Abnormal T-wave forms in cardiac infarction.*—The cove-shaped inverted T wave is commonly seen in cardiac infarction with ABC leads as with standard leads. In addition the cove-shaped upright T wave may also indicate cardiac infarction with leads ABC as with standard leads. Such a case is illustrated in the following. Patient 109 was admitted to hospital with a history of eight hours' duration of sudden severe chest pain passing to the neck and down both arms; he lost consciousness, and on recovery of consciousness was cold and clammy. The ascent of the T wave (Fig. 15) is faster than the descent, it is peaked, and the T wave reaches its maximum after about 50–55% only of the interval from the Q wave to the extreme end of the T wave. Also, it is noticed that the "take-off" is slightly elevated and round-shouldered. This E.C.G. was taken eight hours after the onset.

This type of tracing may be replaced by a more usual pattern later, and in fact five days later this patient's E.C.G. revealed an inverted T wave with well-marked Q wave formation.

(iii) *Electrocardiographic diagnosis of cardiac infarction associated with heart block.*—The diagnosis of cardiac

infarction in the presence of heart block is regarded as a difficult one electrocardiographically. If an earlier tracing free of heart block is available and the E.C.G., taken immediately after symptoms and signs of cardiac infarction appear, shows heart block, this development is generally regarded as due to infarction. Such a case has already been reported with leads ABC (Trethewie, 1953). Right bundle block does not present such difficulty usually, though

FIG. 15.—Cove-shaped upright T wave of cardiac infarction eight hours after onset.

it may be congenital. It was pointed out (Trethewie, 1953) that segment elevation is also evident in such tracings, while in block due to old fibrosis and electrolyte imbalance such segment elevation presents different features. Recently vector-cardiographic work has indicated a feature suggesting infarction associated with block, and this probably depends on the same feature fundamentally. In the present series 17 patients showed varying degrees of block.

There were five cases of left branch bundle block in this series. Three conformed to the pattern seen in hypertension with the gradual production of block following a left strain pattern. In one of these there was no notching of the S wave (first downward complex), but some slurring in leads A and B. In one, notching was present in this complex in A and C, and in one notching in A, B, and C. The B.P. in the first patient was 170/110 on admission. The sedimentation rose and there was a leucocytosis. The history was clinically significant of infarction, and the patient survived.

For comparison the findings in a patient who had no history suggestive of cardiac infarction, but who developed left bundle block associated with aortic regurgitation, are described. The tracing showed slurring only in lead A and not in lead B or C. This patient died two years later, and was shown at necropsy to have aortic incompetence, left ventricular hypertrophy, and some slight general fibrosis without evidence of infarction. Intensive microscopical examination of sections of the heart muscle failed to reveal any evidence of infarction or of sufficiently discrete fibrosis that might have suggested old infarction.

In the other two cases gross notching was present in A. In one the block pattern was also manifest in B and C with gross notching; but in the other leads B and C did not show such a broad biphasic swing pattern. These two, by their elevated segments, clearly indicated infarction. Segment elevation was of the CBa type in one (Patient 36), and the pre-necropsy diagnosis was "infarction: septal, antero-lateral, and slightly on the posterior surface." The standard E.C.G. showed left branch bundle block, which was thought, in view of the history, to be due to infarction but

not conclusive of itself. The patient died 13 days after admission, and the infarct was found to be extensive and in the zones described. In the other (Patient 52) the left branch bundle block was described as atypical, leads B and C not showing the big biphasic swing, and the E.C.G. diagnosis was "antero-lateral infarction: B type, with old posterior infarction." At necropsy, six days later, a yellow gelatinous lamina of infarction was present in the anterior wall of the left ventricle towards the apex. A thrombus was present in the anterior descending branch of the left coronary artery a little below its origin, and there was also another complete occlusion of a small branch of the anterior descending branch. Further details of the distinction between block due to infarction and block due to other causes will form the substance of a separate paper.

Right bundle block patterns were seen in eight instances. Five conformed to a classical infarct pattern (Fig. 16) (Patient 29). This report read: "Old infarct (B) and recent infarct (C): recent septal infarction extending posteriorly with old infarction." At necropsy, 11 days later, "the left ventricle showed extensive recent infarction from the apex into the posterior wall of the left ventricle and anteriorly into the interventricular septum. Between the upper limits of this infarct and the AV junction there was evidence of older infarction." Fig. 17 shows the standard lead findings for this subject. The remaining three patients showed a more marked biphasic swing in the tracings, but segment elevation was present in lead A suggestive of infarction. Two of the latter patients died, and a necropsy was carried out on one. This showed the presence of septal infarction encroaching on the posterior and also slightly on the anterior surface of the heart. Of the former five, four died within a fortnight of the attack. The other patient was discharged on September 14, 1955, and has not returned to hospital. In one, necropsy was not performed; in the other three, infarcts were found—respectively, in the apex of the left ventricle and septum (Patient 22) in the whole of the apex of the left ventricle extending to the base of the septum (Patient 19); and extensively in the posterior wall of the left ventricle and extending anteriorly into the interventricular septum (Patient 29).

Two further patients showed intermittent right bundle block with classical signs of infarction in leads A and C.

FIG. 16.—ABC-E.C.G. showing right bundle block due to cardiac infarction. Necropsy confirmation.

FIG. 17.—Standard E.C.G. of patient whose ABC-E.C.G. is shown in Fig. 16.

Both died, but necropsy was possible on only one, and extensive infarction was detected in the anterior part of the septum and the anterior surface of the left ventricle. In another patient (62) associated with acute ischaemia without actual infarction, the pattern in the standard leads appeared as partial bundle block, but in the ABC leads was associated with RSR' wave formation and slight segment elevation consistent with the diagnosis of acute ischaemia.

One patient showed Wenckebach's phenomenon, and one complete heart block. The former was recognized as posterior cardiac infarction with involvement of the septum. Also, in view of the upright Q in lead B (Trethowie, 1953),

FIG. 18.—Wenckebach's phenomenon: varying PR interval with upright Q in lead B. Posterior cardiac infarction with septal patency. Further details in text.

it was suggested there might also be patency of the septum, but because of the varying block the timing of this wave was not so decisive (Fig. 18), although the T wave indicated that it occurred at the time of the Q wave. At necropsy posterior cardiac infarction was present, the septum was involved, and there was a patency in the infarct in the septum admitting the tip of one finger. The coronary arteries were perfused with radio-opaque material and a block was demonstrated in a large branch of the right coronary artery. The latter patient showed complete heart block, and the E.C.G. report (leads ABC) was "posterior cardiac infarction with involvement of the septum." Necropsy examination confirmed the presence and site of the infarct, and radio-opaque perfusion, carried out as before, showed a similar vascular occlusion.

Theoretical Considerations (Standard Leads)

The action current produced by cardiac muscle as recorded by the intracellular electrode is largely a wide monophasic

FIG. 19.—Intracellular E.C.G. obtained from beating toad heart. Potential: 5 mV. Time base: 0.2 sec.

potential pattern (Woodbury, Hecht, and Christopherson, 1951). We record such a pattern in Fig. 19, a tracing obtained from a toad heart. Kossmann (1954) pointed out that the E.C.G. as recorded in standard leads appears as an approximation of the first derivative ($\frac{dy}{dx}$) of the intracellular action current curve. Nevertheless this should yield an

inverted T wave, which occurs only in some leads. The phenomenon is probably due to the fact that excitation of the heart arises from the bundle tissue in an orderly fashion, the course of the bundle being downwards to the left and forwards. Fig. 20 represents recurring intracellular potentials in the toad heart (1, above), and the tracing (2, below) was obtained from lead C. The changed slope in the intracellular action current may be due to leakage of potassium from the microtip to the muscle fibre investigated, which develops slowly as the preparation remains, while early it conforms to the pattern of Fig. 19. Thus the S-T segment of the E.C.G. (lead C) is isoelectric, corresponding to the horizontal phase of the intracellular trace. Electrical fields are produced by the action currents, and points R (right chest at the base) and X (xiphisternum) develop potentials dependent partly on their distance from the source. The cardiac potentials decrease more rapidly, however, than theory suggests (Vanremmoortere, 1956). Bipolar electrodes R, X (for example, lead C, 2, Fig. 20), simply record potential differences (dy) at their sites at any one instant; changes in successive instants of time (dx) then indicate

FIG. 20.—Intracellular E.C.G.: (1) with corresponding lead C, (2) obtained from beating toad heart.

Pot. $(R - X) = \frac{dy}{dx}$. This is an oversimplification owing to the

multiplicity of heart motor units, but it does indicate the trend of the phenomenon. In the heart, multitudinous action currents are producing varying fields, which are manifest at any one instant by the recorded potential at that time, and there is a specific direction of spread as the successive motor units are excited. Recurring potentials are produced at various points throughout the body—for example, $*P_{(R)}$ at the right arm Einthoven terminal, $P_{(L)}$ at the left, and so on. The structure of the E.C.G. then forms a PQRS complex of approximately similar form from wherever it is recorded on the body surface from any two points, and even when recorded from the developing heart of the chicken. In certain instances the T wave is inverted, depending on the axis chosen for the leads.

Einthoven (E_1) lead 1 then becomes $P_{(R)} - P_{(L)}$

(E_2) lead 2 becomes $P_{(R)} - P_{(F)}$

(E_3) lead 3 becomes $P_{(L)} - P_{(F)}$

Simple arithmetic yields, $E_1 - E_2 + E_3 = P_{(R)} - P_{(L)} - P_{(R)} + P_{(F)} + P_{(L)} - P_{(F)} = 0$
 $\therefore E_1 + E_3 = E_2$

Thus lead E_2 yields no further information than is already contained in E_1 and E_3 , and is therefore redundant. Einthoven

*P refers to the potential change occurring throughout the cardiac cycle.

Since we know ϕ_1, ϕ_2 , see (p) above, and the denominator of the fraction contains terms we know, $\therefore Q$ is known. Thus the moment (strength) of the dipole is known. Thus leads ABC define the dipole completely.

1. *Modification owing to the fact that the emanation is not precisely that of a dipole.*—Craib (1927) has presented strong evidence that we may regard the field as being produced by a dipole, and, since in the final analysis related ionic migration through muscle membrane is associated with the action current produced, this would appear to be a sound hypothesis. Let us first assume it is a point source at the centre producing an emanation of the type $f(t)$. The above treatment still holds, since we can select any particular instant in the cycle of $f(t)$ which will correspond to a selected dipole as treated above.

2. *The thorax is (a) not a sphere, (b) not a perfect conductor and impedance varies throughout its volume, and (c) the heart is not a point source at the centre.*—(a) If points M, L, and R (ϕ_1, ϕ_2, ϕ_3) are not at equidistance from the centre and the thorax forms an ellipsoid since these variations are known, then the treatment is more complicated; but the actual conclusion—namely, that neither the strength (Q) nor the direction of the dipole (l, m, n) can be changed without producing a reflected change in lead A, B, or C—is a reasonable deduction. (b) This factor is difficult to evaluate. Kaufman and Johnston (1943) consider that variations of this nature, since they are not major, do not affect the recorded potentials greatly. (c) Though the heart is not a point source it is still theoretically possible to treat all the emanating points individually and summate algebraically, and therefore we should still obtain an equation related to the known findings appearing from leads A, B, and C. The final difficulty will arise if and when fields produced can annul one another as in precisely accurate phase difference of 180 degrees. This could not be expected to occur with infarction owing to the type of firing off of the heart muscle, and this has already been discussed.

Summary

A series of 161 cases of cardiac infarction on whom ABC electrocardiograms were performed has been presented. Necropsies were done on 36 of these subjects.

The ABC display detected all cases of infarction detected by the present standard leads, and in addition detected infarction not demonstrated by them.

The correlation of infarct site with pattern changes in the ABC leads was determined theoretically and confirmed by necropsy studies.

E.C.G. patterns indicating cardiac degeneration short of discrete infarct formation, with one necropsy study, are described.

Abnormal T-wave form associated with infarction is illustrated.

Heart-block patterns associated with infarct formation are described and presented.

A theoretical analysis of the significance of the Einthoven, Goldberger, Wilson, and ABC leads is presented.

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INCIDENCE OF CORONARY ISCHAEMIA AFTER PARTIAL GASTRECTOMY

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There are many studies relating coronary artery disease to various aspects of lipid metabolism. It has been shown that the incidence is high where the diet contains a large quantity of animal and saturated fat, and low where fat intake is poor (Keys, 1956). Following partial gastrectomy of the Polya type many patients lose weight and most are below their expected weight (Anderson *et al.*, 1955). This is probably due to a low calorie intake and post-gastrectomy symptoms, but impaired digestion and absorption of fat may be a contributory factor. Recent work in gastrectomy patients with ^{131}I labelled fats has shown increased fat excretion and diminished absorption after fatty meals and that fat loss is greater after the Polya operation than after the Billroth I (Everson, 1954; Shingleton *et al.*, 1957). This impaired fat absorption could be reflected in a lower incidence of coronary artery disease in gastrectomy patients.

Material

Of 53 patients who had undergone the Polya type of partial gastrectomy 40 were traced and examined. All were males between 45 and 65 years who had been operated on for severe duodenal ulcer symptoms. The mean lapse of time from operation to interview was 9 years 10 months (range: 7 years 1 month to 13 years 4 months). At the same time, 40 men in the same age group with long-standing symptoms of duodenal ulcer (mean duration 17 years) were studied; 15 of these patients had suffered major complications such as perforation and haemorrhage. As a further control group we examined 40 "normal" males without ulcer or cardiac symptoms admitted for operative treatment of minor surgical and orthopaedic conditions (hernia, varicose veins, haemorrhoids, etc.). In the latter two groups the last five or six patients were selected on an age basis to ensure that the mean ages of each group were closely related (Table I).

Methods

A clinical history and an examination of each patient were undertaken; height, weight, pulse rate, and blood pressure were recorded and a sample of venous blood was withdrawn for haemoglobin and lipoprotein estimations. Electrocardiography was performed, using a direct writer ("mingograph") before and after Master's (1950) double two-step test. The amount of exercise in this test is graduated according to the patient's weight and age. One man