

THE INFLUENCE OF FEAR ON THE ELECTRO-CARDIOGRAM

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While investigating the effect of an anæsthetic on the cardiac action, we chanced on the observation that fear of an impending operation produced remarkable changes in the electrocardiogram of many persons with normal hearts. We therefore proceeded to a systematic investigation in a larger number. Having reported some of these results (Mainzer and Krause, 1939), we are now adding further material, and trying to discuss the cardiographic and clinical significance of the findings.

The circulatory response of the organism, whether normal or pathological, to various psychic stimuli is a wide field of research that has been exhaustively investigated; and the influence of a psychic emotion, such as fear, on the cardiographic tracing forms only a small part of it. Nevertheless, there is such a striking parallelism between our electrocardiograms and the tracings obtained in coronary insufficiency or in myocardial damage that a discussion seems justified.

The electrocardiogram as induced by psychic emotion has been investigated by psychologists (Astruck, 1923 ; Landis and Slight, 1929 ; Weinberg, 1923). The results, which are mostly reported in the archives of psychology or psychiatry—including the paper of Blatz (1925), who is the only one to have studied the influence of fear on the electrocardiogram—are unfortunately not at our disposal. Bier (1930) found high P, R, and T waves after pleasant excitement in some of his experiments. The majority of workers used hypnosis to provoke emotional excitement.

Boas and Goldschmidt (1930), recording the pulse rate previous to and during surgical operations with Boas' cardiometer, found it increased in frequency just before operation and instantly slowed down on the induction of general anæsthesia.

METHOD OF INVESTIGATION

The following procedure was taken in our examinations. In patients of the surgical or gynæcological departments of our hospital we recorded electrocardiograms : (1) one day before operation, the patient knowing nothing of the

impending operation ; (2) on the operating table just before the induction of general anæsthesia ; (3) while under anæsthesia ; and (4) on the day after operation or later, using an amplifier-electrocardiograph. The patients were recumbent, lying flat on their backs, and no drugs were given previous to the taking of the cardiogram. None of them was suffering from valvular disease of any kind or from any clinical symptom of congestive failure. We did not, however, exclude those patients who suffered from coronary sclerosis or arteriosclerotic muscular lesions. We recorded the tracings in the three classical leads. The standard gauge of amplitude was 1mV.=1.0 mm. The præcordial leads are not suitable for this kind of examination, since even a small displacement of the electrode placed near the heart in a second record may cause considerable modification of the tracing.

RESULTS

We made observations upon 58 persons, but the records of 5 of them could not be used for technical reasons. The findings obtained in the remaining 53 are shown in Table I.

TABLE I
ELECTROCARDIOGRAPHIC CHANGES INDUCED BY FEAR OF OPERATION

	Before Operation				Total	
	Normal Records		Pathological Records			
	M.	F.	M.	F.	M.	F.
No electrocardiographic changes induced by fear of operation . . .	10	15	1	3	11	18
Electrocardiographic changes similar to those often seen in coronary insufficiency	0	12	1	4	1	16
Electrocardiographic changes as above, with P and T becoming larger and pointed also	1	3*	0	3	1	6*

* In one of these cases the change was in the P and T waves only.

The majority were women (40 females to 13 males). Twelve of the patients showed an abnormal tracing one day previous to the operation (rest-electrocardiogram) ; they were all suffering from coronary sclerosis, with the exception of one who was undernourished qualitatively as well as quantitatively (avitaminosis) owing to obstruction of the œsophagus through cancer. In 29 of the 53, four of whom had pathological tracings in the rest-electrocardiogram, the records taken immediately before operation were more or less unchanged. The remaining 24 showed pathologically changed tracings owing to fear of the impending operation.

The changes recorded can be classified into three groups :

(a) Those with changes that are most frequently encountered in coronary insufficiency—the S-T interval depressed below the iso-electric level (as compared

with the rest-electrocardiogram) ; the final wave T flattened or completely disappearing or becoming inverted ; S-T and T being deformed into a concave or convex curve ; more rarely, low voltage of the ventricular complex with notching; or a large Q in lead I or III. All these changes are found in more than one single lead.

(b) Those with changes that are usually met with in persons with neuro-circulatory asthenia or in connection with hyperthyroidism—the P and T waves becoming sharply pointed (as compared with the rest-electrocardiogram) and also showing increased voltage.

(c) Those with the changes described under (a) and (b) combined.

The changes of type (a)—as in coronary insufficiency—were encountered most often, i.e. in 15 patients, 4 of whom had already shown a pathological type of tracing at rest. Isolated changes of type (b)—as in persons with neuro-circulatory asthenia—were only found once. Combination of both types were more frequently present; i.e. in 6 persons, in 3 of whom the curve had already been abnormal previous to this.

As has been observed by Boas and Goldschmidt (1930), in the majority of cases a considerable acceleration of the pulse rate usually takes place previous to operation (see Table II), which, if it is very marked, may result in the fusion of the T with the P wave of the following contraction.

TABLE II
INCREASE OF PULSE RATE INDUCED BY FEAR OF OPERATION

Increase of Pulse Rate (per minute)	Nature of Record before Operation			
	Normal	Pathological	Normal	Pathological
	Without Changes of the Electrocardiogram		With Changes of the Electrocardiogram	
0-10	17	2	5	3
11-20.. ..	2	1	2	1
21-30.. ..	4	1	3	1
31-40.. ..	2	0	3	1
Over 40	0	0	3	2
Total	25	4	16	8

These changes in the tracings, attributed to fear, make their appearance in the younger as well as in the higher age groups, though this might not be expected in view of the fact that in the higher age groups a greater incidence of coronary sclerosis and a tendency towards vasoconstrictor vagal action (Gilbert, 1923) prevail. Table III demonstrates that the younger age groups take their full share in this pathological response.

TABLE III

AGE OF THE 24 PATIENTS WITH CARDIOGRAPHIC CHANGES INDUCED BY FEAR

Age (years)	Before Operation	
	With Normal Records	With Pathological Records
Up to 30	12	1
31-40	2	0
41-50	0	3
Over 50	2	4

ILLUSTRATIVE CASES AND ELECTROCARDIOGRAMS

Some typical examples of the various types of "fear tracings" follow.

Fig. 1 shows a series of tracings of type (a), the rest-electrocardiogram of this case being normal. They are from a woman, aged 27, who was having an operation for appendicectomy.

The records taken on the day before operation (A) showed as the only remarkable feature an M-shape of the ventricular complex and a diphasic T in

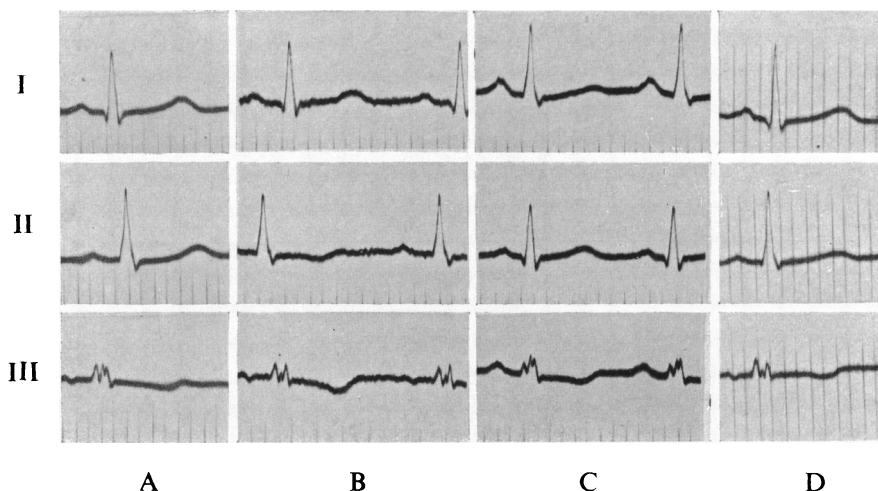


FIG. 1.—Electrocardiograms showing inversion of T_3 and partial inversion of T_2 as a result of anxiety.

(A) at rest ; (B) just before anaesthesia ; (C) during anaesthesia ; and (D) on the day after.

lead III. On the operating table (B) the S-T interval was below the iso-electric level, with a low voltage T in lead II and a negative T in lead III. As soon as general anaesthesia was induced (C) all these changes decreased in intensity. On the day after operation (D) the tracing more or less assumed the shape that had been found on the day before operation.

Fig. 2 shows how the pathological character of a record in coronary insufficiency may become accentuated. It is from a man, aged 70, who was having an operation for removal of the left stellate ganglion for gangrene of the fingers of the left hand. The rest-electrocardiogram of the day before operation (A) showed a slightly negative T_1 and an absent T_2 , with S- T_2 and S- T_3 below the

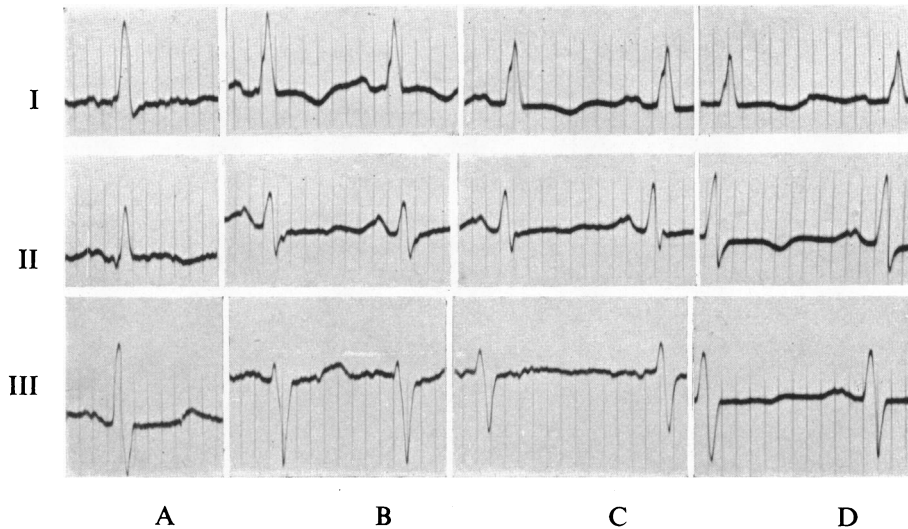


FIG. 2.—Electrocardiograms showing inversion of T_1 and partial inversion of T_2 as a result of anxiety.

(A) at rest ; (B) just before anaesthesia ; (C) during anaesthesia ; and (D) on the day after.

iso-electric level. Immediately before operation (B) the tracing changed ; the ventricular complex became pathological with left axis deviation that was not previously observed, R_2 was much lower, S (of which there was only a trace before) became distinct, and T_1 became negative and T_3 positive. Although in this case too the pathological features induced by fear disappeared to a certain extent during the anaesthesia, the tracing even on the following day was not yet quite identical with the first record.

The only record of our material that may be considered a true representative of type (b) is shown in Fig. 3. It is from a woman, aged 25, who was having an Alexander Adams operation for retroversion of the uterus.

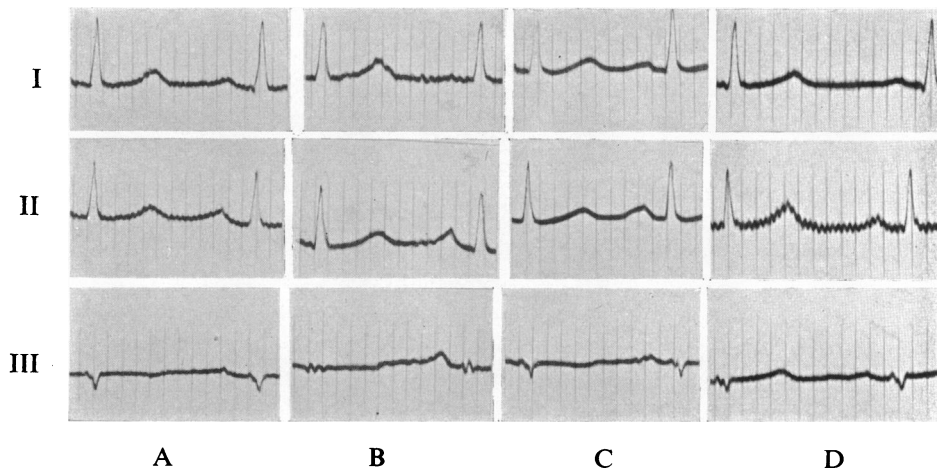


FIG. 3.—Electrocardiograms showing increase of P and T waves as the result of anxiety. (A) at rest ; (B) just before anaesthesia ; (C) during anaesthesia ; and (D) on the day after.

The rest-electrocardiogram (A) showed no pathological features. The ventricular complex showed low voltage in lead III with T_3 negative. Immediately before operation (B) P_2 and even more P_3 increased in voltage and became more sharply pointed. At the same time the ventricular complex was lower, T_3 disappeared, and T_1 and, more markedly, T_2 became higher and more sharply pointed. In this case, too, the changes disappeared to a certain extent during anæsthesia (C). On the day after operation (D) the curve more or less assumed its original shape, save that there was a positive T wave in lead III.

Fig. 4 is characteristic of the combined type (a) and (b), so that the tracing becomes highly abnormal. It is from a man, aged 42, who was having a gastrectomy for a duodenal ulcer with obstruction of the pylorus.

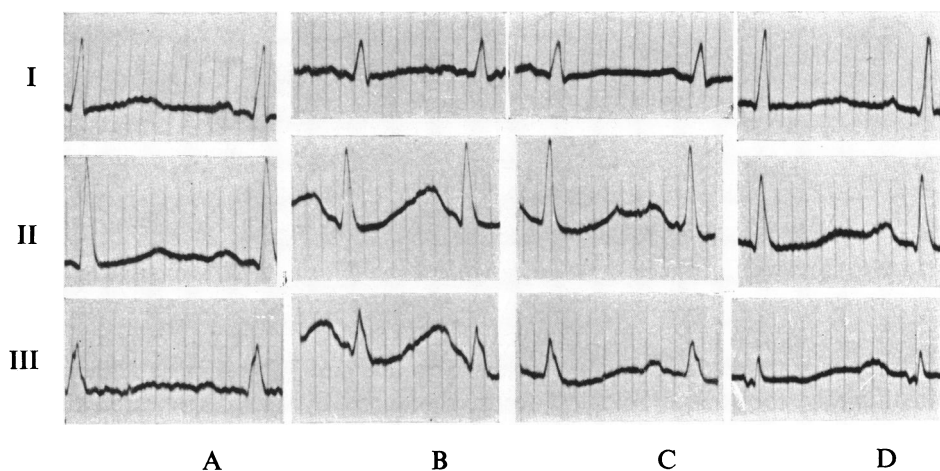


FIG. 4.—Electrocardiograms showing the earlier results combined as a result of anxiety. (A) at rest ; (B) just before anæsthesia ; (C) during anæsthesia ; and (D) on the day after.

The rest-electrocardiogram (A) of this patient may be called normal, T_1 being, however, broad and flat, and T_3 slightly inverted. The shape of this curve may have been influenced by a nutritional disorder (B-complex-avitaminosis). Immediately before the introduction of anæsthesia (B) very marked deformation existed ; in lead I, S-T and T were converted into a broad, slightly convex curve, and in leads II and III, the large T and the subsequent P formed one single large wave. During anæsthesia (C), however, the two deflections became separated to a certain degree. On the day after operation (D) the shape of the record had reverted even further, but not completely to the initial tracing ; P_2 and P_3 remained high and T_3 had become negative.

In all these four cases there was an increased pulse rate before operation.

The question as to how long this "fear-reaction" of the electrocardiogram may persist has been repeatedly touched upon. In most of the remainder (17 of the 20 that showed the "fear-reaction"), a complete return of the curves to normal could be observed on the day following the operation.

The details are shown in Table IV. In 5 of these 25 patients, immediately after the induction of anæsthesia the tracing returned to its original shape. It is just as important, however, that in 3 further cases no return to the original curve took place, even after twenty-four hours.

TABLE IV
DURATION OF THE ELECTROCARDIOGRAPHIC CHANGES INDUCED BY FEAR

	Nature of Records before Operation		
	Normal	Pathological	Total
The deformation disappearing more or less during anæsthesia	1	4	5
The deformation disappearing on the day after operation	13	4	17
The deformation not disappearing completely ..	0	3	3

THE SIGNIFICANCE OF THESE FINDINGS

When taking the records on the operating table just before the induction of general anæsthesia nothing in the proceeding or in the position of the patient differed from that used when taking the other tracings, except for the different psychic condition of the patients ; hence fear of the impending operation can be assumed to be the cause of the cardiographic changes. Moreover, no drugs were given. In five of these cases the elimination of consciousness alone (through anæsthesia) was enough to make the record return to its original shape—a further proof of the emotional origin of the cardiographic changes.

This circulatory response is obviously brought about by way of the autonomic nervous system ; the idea that it may be transmitted in a humoral way only is not compatible with the immediate disappearance of the reaction observed in five of our cases, as soon as the particular psychic strain subsided.

The cardiographic changes of type (*a*) are the same as those found in men (as well as in animals) with coronary insufficiency, and these are also, in part, of a transient nature. Gross anatomical changes concerning the position and the size of the heart or affecting the cardiac walls (pericarditis, myocarditis, etc.) can be excluded from the discussion in view of the transitory nature of the cardiographic alterations. It may, therefore, be assumed that the electrocardiographic fear-reaction of type (*a*) corresponds to a reduced coronary circulation.

The problem of the vasomotor control of the coronary arteries is one of those intricate questions that can only be slightly touched upon here. If we assume (in accordance with Anrep, 1936 ; Wiggers, 1936 ; and Rein, 1931) that in the intact organism the coronary circulation is operated by the vagal tone, we may conclude that a vagal stimulus is responsible for the emotional restriction of the coronary flow. It is very tempting to conclude, on the other hand, that the increased pulse rate—as found in the reaction of type (*b*)—is produced by a sympathetic stimulus ; and that the mixed type of reaction (*c*)

may be brought about by the interaction of both factors. This interpretation remains hypothetical, since the coronary innervation has not yet been fully elucidated. Moreover, the interference of humoral factors cannot be disposed of, especially with reference to reactions of longer duration.

Their Importance for Clinical Cardiography

In clinical cardiography a certain number of records in persons with healthy hearts present a configuration of S-T and T approaching the borderline of the normal or even apparently pathological. Disturbances of internal secretion (ovarian insufficiency, deficiency diseases such as B-avitaminosis, or metabolic disorders) cannot be made responsible for these alterations of the tracing in every instance.

Many of these cases may in fact present fear-reactions, and clinically the fear may either become apparent (fear neurosis) or may remain hidden. Mainzer and Krause (1939) had the opportunity of recording three cardiograms of this kind. It would be a serious error to diagnose an organic heart disease on the grounds of such an abnormal tracing.

Sudden Death from Heart Failure Before and During Operation

Death on the operating table just before the induction of general anæsthesia (Dunbar, 1938) and deaths from cardiac failure during anæsthesia have often been reported. A transitory overdosage of the anæsthetic has been held responsible for the latter occurrence. However, a number of authors, (Hering, 1916), ascribe this event to the state of excitement provoked by the anæsthetic (chloroform) holding a vasomotor reaction (at least that taking place in the peripheral circulation) responsible for these fatal accidents (Alkan, 1930). Our observations, however, make it highly probable that these only represent the extreme cases of the otherwise ordinary fear-reaction, increased by the excitement while under the anæsthetic, and that coronary constriction must be considered the main factor.

Myocardial Damage of Neurogenic (Psychogenic) Origin

A number of clinicians have put forward the view that continuous or repeatedly recurring excitement is likely to advance organic lesions of the cardiac muscle or may even be active in producing them (Klemperer, 1929). This clinical theory of the neurogenic (psychogenic) production of muscular cardiac lesion has been supported to a certain degree by experimental work.

Manning, Hall, and Banting (1937) demonstrated that a prolonged vagal stimulation is able to produce congestion of the capillaries, extravasation of blood, and the development of hyaline foci of degeneration in the myocardium of the dog; and that the occurrence of these phenomena can be prevented by atropine. Even more marked were changes of this kind which Hall, Ettinger, and Banting (1936) were able to induce in the myocardium by administering the vagal substance, acetylcholin, to animals. In older animals this procedure will call forth multiple thromboses within the coronary area and myocardial

infarction as well as foci of hyaline or fatty degeneration with fibrous scar formation. In younger animals the arterial changes are absent and the myocardial damage is less pronounced ; in this case too atropine prevents their occurrence.

When transferring the results of these experiments to human pathology the objection might be raised that in the animal experiment the vagal stimulus and the acetylcholin dosage reached an unphysiological degree. In human pathology, however, similar observations have been made in connection with angina pectoris.

Ordinary angina pectoris is of transient nature, clinically and often electrocardiographically too. It subsides just as quickly as the psychic emotion before operation and the resultant circulatory response. Various investigators during the last decade have shown that, in spite of this transient character, the paroxysm of angina pectoris may give rise to the formation of circumscribed myocardial necrosis, which, in the course of time, may be converted into fibrous scar tissue (Gallavardin, 1932 ; Büchner, 1932 ; Holzmann, 1937). If, therefore, the heart has been subjected to a considerable number of attacks, the myocardium may be riddled with necrotic or, later, fibrous foci of microscopical size. As pointed out above, the vasomotor fear-reaction of the cardiographic type (*a*) shows a perfect analogy with that provoked by the angina pectoris paroxysm. If this is correct, the animal experiment as well as the clinical and pathological findings should lead us to envisage the probability that the emotional vagal reaction may also produce permanent anatomical lesion of the myocardium, the extent of which may depend on the degree and the frequency of the reaction.

Thus, our findings support in a certain degree the clinical hypothesis of a psychogenic origin of organic cardiac diseases.

SUMMARY

On the operating table immediately before induction of general anæsthesia, an abnormal electrocardiographic record was found to develop in roughly two fifths of 53 cases, in comparison with the tracing of the previous day. These alterations were observed in persons with cardiac disorders, where they merely accentuated the pathological character of the cardiogram already existing, and also occurred frequently in patients with normal cardiograms. While in a number of patients the changes disappeared under the anæsthetic, or at least by the next day, they were in some cases still encountered twenty-four hours after operation.

The changes may be classified into three groups :

(*a*) S-T is depressed below the iso-electric level, and T is low, inverted, or absent altogether, S-T and T showing some deformation similar to that appearing in coronary insufficiency ;

(*b*) P and T are high and become sharply pointed, as is also found in neuro-circulatory asthenia ;

(*c*) a combination of the changes quoted under (*a*) and (*b*).

Factors likely to modify the records, other than the excitement owing to fear of the impending operation, can be ruled out. In some patients the curve returns to its original shape even while they are still under the anæsthetic, thus supporting the hypothesis of a fear reaction.

In view of the analogies existing between "fear-electrocardiograms" and other types of tracings, it is assumed that the curves of type (a) are brought about by a reduced coronary flow, mainly to be attributed to vagal stimulation; that sympathetic stimulation is responsible for the development of the curves of type (b); and that type (c) is the result of the interaction of both factors. It is improbable that only humoral factors could be active in bringing about these phenomena, in view of their rapid disappearance.

Thus in clinical cardiography a number of abnormal records that can be explained in no other way probably present genuine fear-tracings, particularly where neurotic persons are concerned.

Death from cardiac failure on the operating table immediately before the induction of general anæsthesia as well as during anæsthesia should, therefore, at least in some cases, be considered as the extreme outcome of an otherwise usual fear-reaction.

The coronary spasms of an ordinary attack of angina pectoris may give rise to the formation of microscopically recognizable necrotic foci in the myocardium. Neurogenic (vagal) lesions of the coronary arteries and myocardium have also been encountered in animal experiment. Thus myocardial damage could be induced by the vasomotor fear-reaction, as becomes apparent in the curves of type (a), and could be attributed to coronary constriction of vagal origin.

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