CHEST LEAD (CR₁) ELECTROCARDIOGRAMS IN AURICULAR FIBRILLATION

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Lewis (1910) was the first to demonstrate the value of chest leads in displaying the movements of the auricle in auricular fibrillation. A trial of five different leads convinced him that those taken over the right auricle showed maximal auricular oscillations and minimal ventricular complexes which facilitated a study of the auricular waves. In a case of complete heart block Cohn and Lewis (1913) found that oscillations from auricular action were greatest when leads were taken directly from the chest wall, the electrodes being placed in the region of the right auricle. Drury and Iliescu (1921) tried two chest leads in 24 patients with auricular fibrillation. In their first or sternal lead an electrode over the second rib on the right side was paired with one over the seventh costal cartilage on the same side, and in their second or antero-posterior lead an electrode at the centre of the sternum was paired with one at the level of the inferior angle of the scapula, two inches to the right of the spine. They favoured the antero-posterior lead. Holzmam (1937) said that abnormalities of the P wave in the electrocardiogram were best recorded by placing the exploring electrode over the right auricle. Lian and Pinchenzon (1938, 1940) used a chest lead with one electrode over the manubrium and the other in the fifth right intercostal space for the investigation of auricular rhythm, and named it the "precordial auricular lead S5." The application of this lead in one case of arrhythmia, which was thought to be auricular fibrillation, gave a tracing exhibiting large auricular waves. They described the condition as auricular tremulation and maintained that it was intermediary between auricular flutter and fibrillation. They postulated that in fibrillation both the duration and shape of the auricular complex in the cardiogram varied, in auricular flutter there was no variation, and in tremulation it was slight. It was while studying the form of the P wave in the chest lead cardiogram CR₁ * in various cardiac disorders that I became interested in tracings obtained from patients with auricular fibrillation. This lead was

* This designation was suggested by the committee reporting on the standardization of precordial leads (*Amer. Heart J.*, 1938, 15, 235). C is an abbreviation for chest, R for right arm, and the subscript 1 denotes the position of the precordial electrode, namely in the fourth intercostal space at the right border of the sternum.

then recorded in 60 patients in whom clinical examination and the limb lead cardiogram had provided the diagnosis of fibrillation.

It is known that the auricular movements in fibrillation are never faithfully portrayed in the limb lead cardiogram ; the tracing may show no oscillations, or if it does the curves are not constant and vary even in the same patient from insignificant waves to prominent ones, these showing the greatest prominence in one or other lead. When the limb lead cardiograms from the 60 patients were examined for auricular waves they were absent in 28 ; in the remaining 32 the curves were better displayed in leads III and II than in lead I. Actually the oscillations were moderately prominent in lead III in 24 cases, in lead II in 20, and in lead I in 10 cases. Even when distinct coarse waves are seen in limb lead cardiograms they are distorted by movements from sources other than the auricle. In the CR₁ cardiogram extraneous somatic waves are more completely eliminated while the ventricular complexes are small, but its chief advantage over the limb leads appears to be its specificity for recording auricular movements, owing to the plane which the lead traverses. Fig. 1-4 and many

FIG. 1.—Case 29. Auricular fibrillation without apparent cause. Female, aged 64. Auricular rate, 375 a minute. In this and some of the other figures dots denote the summits of the auricular waves.

of the subsequent figures illustrate this. None the less, in many patients this chest lead fails to display the auricular wave to much better advantage than the limb lead. Again, the periodic variation in the amplitude of the oscillations which is characteristic of the limb lead tracings may also be a feature of many of the curves obtained when leading direct from the chest.

In this series 39 CR₁ cardiograms showed large auricular waves (Type I) ; in the remaining 21 the waves were not seen to much better advantage than in the limb lead cardiogram (Type II).

FIG. 2.—Case 22. Auricular fibrillation and mitral stenosis. Female, aged 29. Auricular rate, 384 a minute.

FIG. 3.—Case 25. Auricular fibrillation without apparent cause. Male, aged 53. Auricular rate, 375 a minute.

FIG. 4.—Case 32. Auricular fibrillation and mitral stenosis. Female, aged 50. Auricular rate, 362 a minute.

LARGE AURICULAR WAVES IN CR₁: TYPE I

Although 39 of the 60 patients have been included in this group, 2 (Cases 38 and 39) are kept apart from the remaining 37 and will be discussed at the end of the section.

A description of the auricular wave in the CR₁ cardiogram should relate to its *form*, *rhythm*, and *frequency*, and these features of the curve will now be considered separately.

Form of the Auricular Wave

The form of the auricular wave in the CR₁ cardiogram varied from patient to patient, but it remained fairly constant in the same individual with each curve differing slightly from the other. It did not show that regularity of pattern characteristic of the auricular flutter curve, but there was a close resemblance. In most tracings the fibrillation waves presented a gradual upstroke followed by a rather steep downstroke. The curves were written in continuity and without a pause at the iso-electric level, proving that the auricle was at no time electrically quiescent. Further typical illustrations are given in Fig. 5-9. Often the amplitude of the waves became subject to a phasic variation, from time to time increasing and diminishing in height. During a period when the amplitude of the oscillation diminished (see end of CR₁ tracing in Fig. 5) the wave often became bifid, giving the appearance of two

FIG. 5.—Case 17. Auricular fibrillation and mitral stenosis. Male, aged 44. Auricular rate, 394 a minute. Bifid auricular wave at end of CR₁ lead.

FIG. 6.—Case 31. Auricular fibrillation and mitral stenosis. Female, aged 22. Auricular rate, 363 a minute.

FIG. 7.—Case 36. Auricular fibrillation and thyroid toxæmia. Male, aged 55. Auricular rate, 350 a minute.

FIG. 8.—Case 18. Auricular fibrillation and mitral stenosis. Female, aged 25. Auricular rate, 394 a minute.

separate waves. No extrinsic cause for this variation could be made out ; the respiratory movements, for instance, did not influence the amplitude, and the same observation was made by Drury and Iliescu (1921). When comparing five different chest leads, Lewis (1910) stated that the oscillations were conspicuous or the reverse according as the contacts were close to or distant from the right auricle, but that whenever conspicuous oscillations were obtained, then inconspicuous oscillations appearing in part of the tracing were an expression of some intrinsic variation in the auricle. In my cases the amplitude of the fibrillation wave did not depend on the condition initiating the arrhythmia nor on the size of the right auricle, for the waves were no larger in patients with mitral stenosis than in those without, who were seen on radioscopy to have a heart unaffected in shape or in size (Fig. 1 and 3).

Rhythm of the Auricular Wave

Describing the oscillations that replace the normal P waves when auricular fibrillation sets in, Lewis (1925) stated that they were never quite regular in form, amplitude, or length. The 37 patients in this series may be divided into three groups according to the rhythm of the fibrillation waves in the CR₁ electrocardiogram. There were 18 patients in the largest group where the rhythm was irregular ; even in these the irregularity was not obvious at first and only emerged when measurements were taken. It was difficult to determine the influence of the ventricular systole or diastole on the auricular rhythm in these cases. In the second group of 13 patients the rhythm of the auricular

waves was regular except when it was disturbed (occasionally, frequently, or constantly) by ventricular systole; in 7 the auricular wave succeeding ventricular systole was delayed, and in 6 it was hastened. This finding is in disagreement with that of Drury and Iliescu (1921), who stated that both ventricular systole and diastole were without effect on the auricular oscillations. The third group held 6 patients in whom the rhythm of the auricular waves was regular and was undisturbed by ventricular systole; in these patients although the auricular rhythm was regular and very like auricular flutter, the shape of the waves differed slightly from beat to beat.

Frequency of the Auricular Wave

Different rates have been assigned to auricular contraction in auricular fibrillation by different writers. Thus, Battro (1937) mentions 400 to 600

TABLE I
37 PATIENTS WITH LARGE AURICULAR WAVES IN THE CR₁ CARDIOGRAM (TYPE I)

Case No.	Age	Cause of Fibrillation	Digitalis or No Digitalis	Average Ventricular Rate	Auricular Rate	Limb leads showing Fibrillation Waves
1	64	No cause found	No D.	100	488	II and III
2	63	Mitral stenosis	No D.	100	453	0
3	60	No cause found	D.	100	450	II and III
4	43	Mitral stenosis	D.	90	450	0
5	34	No cause found	No D.	85	450	III and II
6	30	Mitral stenosis	D.	37	437	0
7	45	do.	D.	75	436	II and III
8	45	do.	D.	85	428	0
9	39	do.	D.	130	428	II and III
10	33	do.	D.	70	420	I
11	26	do.	D.	30	416	0
12	68	Hypertension	No D.	150	409	II
13	38	Mitral stenosis	No D.	92	400	III and II
14	58	No cause found	No D.	130	400	III
15	63	Hypertension	D.	90	400	0
16	46	Mitral stenosis	D.	60	400	III and II
17	44	do.	No D.	100	394	0
18	25	do.	D.	120	394	II
19	29	do.	D.	55	390	I
20	37	do.	D.	100	388	I
21	76	Hypertension	No D.	75	385	III
22	29	Mitral stenosis	D.	100	384	0
23	66	do.	D.	130	375	II and III
24	53	do.	D.	66	375	0
25	53	No cause found	No D.	85	375	III and II
26	38	Mitral stenosis	D.	85	375	II
27	31	do.	D.	130	375	0
28	39	do.	D.	120	375	I, II, and III
29	64	No cause found	No D.	120	375	III and II
30	51	Mitral stenosis	D.	64	375	II and III
31	22	do.	D.	85	363	0
32	50	do.	D.	150	362	II and III
33	43	do.	D.	75	362	II
34	39	do.	D.	72	355	0
35	27	do.	D.	66	353	I and III
36	55	Thyroid toxæmia	No D.	130	350	III and II
37	49	Mitral stenosis	D.	75	350	I

and Bramwell (1932) 500 to 600. Drury and Iliescu (1921) found the auricular rate to be 390 to 588 in twenty-four cases (chest leads), with an average of 473. Herrmann (1936) stated that it was 450 to 600, Levine (1936) 400 or more, Lewis (1925) rarely below 400 and rarely above 600, Pardee (1928) 350 to 500, Wenckebach and Winterberg (1927) 434 in one patient with chest lead, and White (1937) gave the figure as 400 to 600. The disparity in the figures is explained by the fact that the observations have been based in most instances on the frequency of the auricular waves in the limb lead cardiogram. Such observations are necessarily precarious, owing to the imperfections of this electrocardiogram in showing the complete details of auricular contraction. Thus, in calculating the rate of the main movement of the auricle both the coarse and the fine oscillations have been separately regarded as depicting auricular contraction. This impression of greater frequency created by notching or splitting of the main auricular wave is illustrated towards the end of the CR_1 tracing in Fig. 5. Again, even if the oscillations in the limb lead cardiogram are moderately distinctive for a moment calculation of the auricular rate based on a few beats will be inaccurate. Table I shows the remarkable constancy of the rate of auricular contraction in patients with auricular fibrillation.

FIG. 9.—Case 9. Auricular fibrillation and mitral stenosis. Female, aged 39. Auricular rate 428 a minute.

FIG. 10.—Case 38. Mitral stenosis. Male, aged 43. Auricular rate, 312 a minute. Limb leads suggest auricular fibrillation. The CR_1 lead shows auricular flutter (see text p. 254).

More than half had an auricular rate of 375 to 400 a minute ; with two exceptions the rate was 350 to 450, and the average rate was 400 for the 37 patients. The auricular rate did not depend on the ventricular rate, for a patient showing the highest auricular rate (488) had an average ventricular rate of 100, while a patient with the lowest auricular rate (350) had a ventricular rate of 130. Digitalis was found to have no effect on the auricular rate in patients re-examined with the CR₁ electrocardiogram during a period of digitalis therapy. Quinidine did slow the auricular rate (see Fig. 12).

Two patients (Cases 38 and 39) were regarded clinically as auricular fibrillation and mitral stenosis. In Case 38 the limb lead cardiograms supported this view, although the tracing in some ways resembled flutter : the CR₁ cardiogram, however, gave the diagnosis of auricular flutter (Fig. 10). In Case 39 the ventricular complexes were infrequent and usually irregular, and no auricular waves were visible ; in all, 15 limb lead cardiograms were recorded, and they appeared to support the diagnosis of auricular fibrillation. The CR₁ cardiogram still left doubt about the exact diagnosis of the type of auricular activity but showed 2 : 1 auriculo-ventricular block (Fig. 11). This last tracing shows a rhythm similar to that found in chest lead cardiograms in paroxysmal tachycardia, which are being investigated.

FIG. 11.—Case 39. Mitral stenosis. Female, aged 52. Limb leads suggest auricular fibrillation, but CR₁ shows that whatever the auricular rhythm may be there is 2 : 1 auriculo - ventricular block. Auricular rate, 210 a minute.

FIG. 12.—Slowing of the auricular rate by quinidine. The tracing above was taken before, and that below after, a week on quinidine (9 grains daily). The auricular rate was reduced from 375 to 340 a minute.

SMALL AURICULAR WAVES IN CR₁ : TYPE II

In most of the 21 patients of this group the CR₁ cardiogram demonstrated auricular waves which were more conspicuous than those recorded in the limb lead cardiogram, but the curves were neither prominent nor constant enough to permit a study of the form of individual waves nor to allow an

estimate of the rate of auricular contraction. An attempt was made to discover some factor that might determine whether a patient with auricular fibrillation would show large (Type I) or small (Type II) auricular movements in the CR₁ cardiogram. To this end special attention was paid to the cause and duration of the arrhythmia, the ventricular rate, digitalis influence, the size of the right auricle, presence of "fibrillation" waves in the limb lead cardiogram, thickness of the chest wall, and to the possibility of a faulty technique in applying the chest electrode.

The *cause* of the arrhythmia appeared to have no influence in deciding the amplitude of the auricular wave in the CR₁ cardiogram. In the favourable series (Type I) of 37 patients, mitral stenosis was the cause in 27, hypertension in 3, thyroid toxæmia in 1, and cause unknown in 6. In the unfavourable series (Type II) of 21 patients, mitral stenosis was the cause in 18, and the etiology was unknown in 3.

An attempt was made to ascertain the *duration* of the fibrillation in each patient. From this inquiry the prominence of the auricular wave did not appear to depend on the duration of fibrillation; the arrhythmia appeared to be recent (within three months) in 14 of 37 Type I cases and in 6 of 21 Type II cases.

TABLE II

21 PATIENTS WITH SMALL AURICULAR WAVES IN THE CR₁ CARDIOGRAM (TYPE II)

Case No.	Age	Cause of Fibrillation	Digitalis or No Digitalis	Average Ventricular Rate	Limb leads showing Fibrillation Waves
1	60	No cause found	No D.	60	0
2	48	Mitral stenosis	No D.	85	III and I
3	39	do.	D.	60	0
4	34	do.	D.	80	0
5	55	do.	D.	94	0
6	21	do.	No D.	42	0
7	54	do.	D.	60	0
8	44	do.	D.	55	0
9	51	do.	D.	50	0
10	45	do.	D.	85	0
11	39	do.	D.	42	0
12	58	do.	D.	90	0
13	48	do.	D.	50	0
14	42	do.	D.	55	0
15	48	do.	D.	90	III and I
16	68	No cause found	D.	85	III
17	60	do.	D.	85	0
18	34	Mitral stenosis	D.	42	I and III
19	58	No cause found	No D.	66	III
20	58	Mitral stenosis	D.	46	0
21	44	do.	D.	60	III, II, and I

When the *ventricular rate* was compared in patients in the two groups a difference was observed. It was much more rapid in those with prominent auricular waves, averaging 92 per minute for the 37 patients (see Table I), compared with 66 for the 21 patients with inconspicuous waves (see Table 2).

Again, whereas no Type II patient had a rate higher than 94, in Type I patients it was over 100 per minute in 17. Nevertheless, although conspicuous auricular waves were always present when the ventricular rate was rapid, they were sometimes observed with a moderately slow ventricular rate (Fig. 3).

The inquiry on rate led to an investigation of the incidence of *digitalization* in patients in each group. Although it was a little commoner in Type II patients, the influence of digitalis on the auricular wave was not obvious.

It was natural to assume that in Type I patients a projection of the *right auricle* beyond the right border of the sternum favoured the recording of auricular movements by an electrode placed in this area. Lewis (1910), when advocating the more frequent use of chest leads in the study of oscillations produced by the fibrillating auricle, stated that the best curves were obtained in cases in which the right auricle was distended, as in mitral stenosis, when a large part of its surface lay in contact with the chest wall. The present investigation does not support this view, for it showed that conspicuous auricular waves in the CR₁ electrocardiogram did not depend on extension of the heart to the right. Thus, most of the patients were examined by radioscopy, and in 15 patients showing conspicuous waves enlargement of the right auricle was either absent or slight, whereas in 9 patients showing inconspicuous waves right auricular enlargement was either moderate or considerable.

That patients with prominent fibrillation waves in the limb lead electrocardiogram would also show conspicuous waves in the CR₁ was expected, and this proved to be the case, so that coarse auricular oscillations were seen in one or more limb leads in 25 of the 37 Type I cases and they were absent in 15 of the 21 Type II cases. At the same time the exceptions emphasize that this criterion is not a constant guide in predicting the two types.

The thickness of the chest wall did not affect the amplitude of the auricular waves in the CR₁ electrocardiogram, and that inconspicuous curves were not the result of a faulty technique in applying the chest electrode was proved by recalling patients of both series and obtaining in them tracings identical with the previous records.

SUMMARY AND CONCLUSIONS

The chest electrocardiogram, CR₁, was taken in 60 patients with auricular fibrillation in order to study the auricular wave. Large and conspicuous waves, resembling those of auricular flutter, were found in 39 patients (Type I). In the remaining 21 cases (Type II) the auricular waves were small and no more conspicuous than in many limb lead electrocardiograms.

No factor was discovered that determined in a patient with auricular fibrillation whether the auricular movements would be represented in the CR₁ cardiogram by large or by small waves, except that if the ventricular rate was high and the auricular oscillations were conspicuous in the limb lead cardiogram, distinct waves were likely to show in the CR₁ cardiogram. The ætiology and the duration of the arrhythmia, the size of the right auricle, digitalization

or its absence, and the thickness of the chest wall, did not seem to decide whether a patient would exhibit large (Type I) or small (Type II) auricular waves.

The features of the auricular waves were clearly shown in those patients classified as Type I, and these were specially studied. The *form* of the wave, varying slightly in different patients, resembled the waves found in auricular flutter, although lacking the same constant pattern. The waves recurred continuously without a pause at the iso-electric level, and each showed a gradual upstroke followed by a steeper downstroke. The amplitude of the wave was occasionally subject to a natural variation, and this was independent of any known extrinsic factor, including respiration. Small waves were often bifid, thereby giving the appearance of two waves and explaining the more frequent oscillations seen in the limb lead cardiogram. The wave was no larger in mitral stenosis than in non-rheumatic auricular fibrillation.

The *rhythm* of the auricular waves was slightly irregular in 18 patients, regular in 13 except for interruptions by ventricular systole when the succeeding auricular wave was as often delayed as quickened, and quite regular in 6 cases, where the rhythm was undisturbed even by ventricular systole, so that the tracing closely resembled that of auricular flutter.

The *frequency* of auricular waves in auricular fibrillation was found to be reasonably constant; the rate was 375 to 400 per minute in more than half the cases; with two exceptions the range was 350 to 450 and the average rate for all patients was 400. Higher rates of auricular contraction generally assigned to auricular fibrillation have been based on observations on limb lead cardiograms which often contain more than one oscillation representing a single beat of the chest lead cardiogram. The rate was uninfluenced by digitalis but it was reduced by quinidine.

In two patients in whom a clinical diagnosis of auricular fibrillation was supported by a limb lead cardiogram, the CR₁ tracing demonstrated auricular flutter in one and 2 : 1 auriculo-ventricular block in the other. The value of this chest lead is also seen in elucidating cases of so-called impure flutter, better regarded as fibrillation, and in the interpretation of records with auricular waves in the limb lead cardiogram sometimes subdued or obscured, as in heart block and in paroxysmal tachycardia.

The chest, CR₁, cardiogram permits the limited definition of *auricular fibrillation* as a succession of waves at a rate of about 400 per minute, differing slightly in size and shape, and with a rhythm almost as often regular as irregular. The ventricular responses in fibrillation have no constant relationship to the auricular waves, probably because the auricular rate is too high for any regular control.

In contrast *auricular flutter* shows a succession of waves at a rate of about 300 per minute, identical in size and shape, and perfect in rhythm. The ventricular responses in flutter are determined by the auricular waves giving a fixed or varying ratio.

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