ŒSOPHAGEAL ELECTROCARDIOGRAMS IN AURICULAR FIBRILLATION *

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A critical study of electrical events occurring in the posterior portions of the human heart has been made possible by the use of œsophageal leads in clinical electrocardiography (Cremer, 1906; Lieberson and Liberson, 1934; Luisada, 1935; Brown, 1936*a*, 1936*b*; Hamilton and Nyboer, 1938; Spühler, 1938; Nyboer, 1939; Deglaude and Laubry, 1939). The œsophagus lies close to the posterior surface of the left auricle, being separated from it by a space, 0.5–1 cm. wide, composed of areolar tissue and by the two layers of the pericardium. The right auricle lies 4–6 cm. anteriorly, across the cavity of the left; but beneath the most inferior part of the left auricle the œsophagus is separated from the inferior vena cava and the right auricle by a distance of only 1–2 cm.

Brown (1936*a*) showed that an electrode lying in this part of the œsophagus provided a nearly direct lead for the surface of the left auricle and, when paired with a distant electrode, was capable of recording faithfully the electrical changes of this region. From his study of 21 patients exhibiting auricular fibrillation Brown (1936*b*) concluded that, when it has become established and is not liable to spontaneous reversion to normal rhythm, the auricular deflections recorded by the œsophageal lead are of small amplitude and irregular form and do not exhibit intrinsic deflections, although the electrode lies against or close to the posterior (left auricular) segment of the pathway round which a circulating mother impulse is supposed to travel. Deglaude and Laubry (1939) also failed to record intrinsic deflections. Brown, however, did obtain records showing intrinsic deflections from two patients in whom fibrillation was associated with thyrotoxicosis; in one of these normal rhythm appeared after thyroidectomy. He suggested that, when auricular intrinsic deflections are recordable by the œsophageal lead, fibrillation may not be firmly established and may be susceptible to spontaneous reversion.

We decided therefore to examine by means of the œsophageal lead cases of auricular fibrillation in which the arrhythmia was known to be paroxysmal or

* The results of this study were presented in abstract before the Edinburgh Pathological Club in March, 1939.

those in which normal rhythm was restored by quinidine, and to compare the resulting curves with those obtained from cases of established fibrillation.

The method employed, modified from that of Brown, has been described (Hamilton and Nyboer, 1938). Records were obtained in each case from several auricular levels as well as from supra- and infra-auricular sites. Repeat œsophageal cardiograms were always made from the same levels as the original records except where otherwise stated in the figures.*

Nine patients exhibiting auricular fibrillation were studied. Reversion to normal rhythm occurred in four, quinidine having been given to three of these, and in the other five fibrillation persisted.

I. CASES THAT REVERTED TO NORMAL RHYTHM

In all four the auricular deflections recorded by the œsophageal lead were of greater amplitude than those in the standard lead tracings.

Three cases where auricular intrinsic deflections were recorded

Case 1 was that of a female, aged 35, suffering from chronic rheumatic mitral disease; she had had short attacks of dyspnœa for fifteen years, paroxysmal fibrillation having been recorded several times during the previous three years. Digitalis was given for the relief of congestive cardiac failure, but no quinidine.

Cardiograms (Fig. 1 B) by standard leads showed auricular fibrillation with coarse "f" waves having a maximum amplitude of 0.3 mv. In the œsophageal cardiograms † numerous auricular intrinsic deflections were seen, often in pairs or groups separated by smaller, irregular, slurred waves. A normal curve is shown for comparison in Fig. 1 A. The maximum amplitude of the auricular deflections was 0.6 mv. and the minimum less than 0.1 mv. After reversion to sinus rhythm normal œsophageal curves were obtained.

Case 2 was that of a male, aged 48 years, suffering from coronary arterial disease and chronic alcoholism. Fibrillation started two weeks before admission with sudden palpitation, dyspnœa, and œdema which was not relieved by full digitalization; salyrgan, however, caused marked diuresis and the patient lost 43 lb. of weight in seven days with clinical improvement. Fibrillation was still present five weeks later. Normal rhythm was restored when 1.0 g. of quinidine sulphate had been taken in two days, and remained for a further six months during which he took no quinidine.

Standard lead cardiograms (Fig. 1 C), made before quinidine was given, showed fibrillation with low voltage "f" waves having a maximum amplitude of 0.2 mv. Œsophageal cardiograms contained many auricular intrinsic deflect-

* Since the work recorded in this paper was carried out the joint recommendations of the American Heart Association and the Cardiac Society of Great Britain and Ireland relating to the method of recording præcordial and other semi-direct leads have been generally accepted. We have therefore reversed all œsophageal lead curves in this paper so that they conform in appearance to those which may be obtained by the new method of leading.

† In this and all subsequent descriptions the term "œsophageal cardiogram" is used to refer to the tracing obtained when the electrode lay at the level of the auricles. The ventricular œsophageal electrocardiogram is not considered in this paper.

FIG. 1.—(A): Standard and æsophageal lead cardiograms of a normal male, aged 30 years, for comparison. In this and the other figures the distance of the mid-point of the æsophageal electrode from the incisor teeth is recorded. String sensitivity, 1 mv. = 10 mm. Time-marker, 0.2 and 0.04 second.

(B)–(E): Auricular intrinsic deflections in æsophageal cardiograms.

(B) Case 1; (C) Case 2; (D) Case 3: auricular fibrillation; subsequent normal rhythm.

(E) Case 5, (F) Case 6: auricular fibrillation; no return of normal rhythm.

ions; they occurred in groups separated by smaller, irregular, slurred waves, and had a maximum amplitude of 0.8 mv. and a minimum of less than 0.1 mv. After normal rhythm was restored they were of normal form.

Case 3 was that of a male, aged 53, suffering from arteriosclerosis and coronary arterial disease; he had complained of dyspnœa on exertion for one year. Rhythm was normal, apart from occasional supraventricular extrasystoles, until the tenth day after admission, when the auricles started to flutter at a rate of 300 per minute with a 2 : 1 A-V ratio. Fibrillation was induced by

digitalis and persisted for ten days, after which quinidine sulphate was given along with the digitalis; after 1.8 g. had been taken in two days normal rhythm reappeared and remained.

Fibrillation with small "f" waves, having a maximum amplitude of less than 0.1 mv., was shown in the standard lead cardiograms (Fig. 1 D) taken before quinidine was given. In the œsophageal cardiograms numerous auricular intrinsic deflections were seen, occurring in groups separated by smaller, irregular, slurred waves; they had a maximum amplitude of 0.7 mv. and a minimum of 0.2 mv.; after the reappearance of normal rhythm they were of normal form.

One case where no auricular intrinsic deflections were recorded

Case 4 was that of a male, aged 56, suffering from arteriosclerosis and coronary disease. Paroxysms of fibrillation had been recorded repeatedly during the preceding four years, and had usually ceased spontaneously though digitalis had often been given. The paroxysm observed by us lasted for two days and ended after 0.2 g. of quinidine sulphate; no digitalis had been given.

During the paroxysm standard lead cardiograms (Fig. 2 A, see page 267) showed fibrillation with small "f" waves having a maximum amplitude of 0.1 mv. In the œsophageal cardiograms the auricular deflections were very irregular in size and form, being only slightly larger than the "f" waves of the standard lead curves. Although many feet of record, obtained from various auricular levels, were studied, no certain instance of intrinsic deflection could be found. The auricular deflections had a maximum amplitude of 0.3 mv. and a minimum of 0.1 mv. Normal œsophageal curves were secured after normal rhythm reappeared.

II. CASES THAT DID NOT REVERT TO NORMAL RHYTHM

In all five the auricular deflections recorded by the œsophageal lead were of greater amplitude than those in the standard lead tracings.

Two cases where auricular intrinsic deflections were recorded

Case 5 was that of a female, aged 46, suffering from chronic rheumatic mitral disease and an acute exacerbation. She had had dyspnœa on exertion for six years. The presence of fibrillation had been recorded ten weeks before the observed hospital period during which the arrhythmia was continuously present. She received digitalis but no quinidine.

Standard lead cardiograms (Fig. 1 E, see page 265) showed fibrillation with "f" waves of moderate size having a maximum amplitude of 0.2 mv. In the œsophageal cardiograms the auricular complexes were of varying amplitude, some smaller and some larger than those in the standard lead curves. A few auricular intrinsic deflections were recorded, but these tended to occur singly and were separated by completely slurred waves; the maximum amplitude was 0.5 mv. and the minimum less than 0.1 mv.

Case 6 was that of a male, aged 79, suffering from arteriosclerosis and coronary disease. He had had increasing dyspnœa on exertion for one year

and œdema of the ankles for five weeks. There was no definite episode suggesting the onset of the auricular fibrillation, which remained throughout his 18 days in hospital. He received digitalis but no quinidine.

Standard lead cardiograms (Fig. 1 F) showed auricular fibrillation with small "f" waves having a maximum amplitude of 0.1 mv. In the œsophageal curves numerous auricular intrinsic deflections were seen, occurring in groups between which were smaller, irregular, slurred waves; they had a maximum amplitude of 0.45 mv. and a minimum of less than 0.1 mv.

Three cases where no auricular intrinsic deflections were recorded

Case 7 was that of a male, aged 76, suffering from arteriosclerosis and coronary disease, who had complained of dyspnœa on exertion and œdema of the ankles for two years. Throughout this period auricular fibrillation was known to be present. He was given digitalis but no quinidine.

Standard lead cardiograms (Fig. 2 B) showed fibrillation with small, often imperceptible "f" waves having a maximum amplitude of less than 0.1 mv. In the œsophageal tracings the auricular waves were also small, though larger than those in the standard lead curves, having a maximum amplitude of slightly over 0.1 mv. Although records were made from many auricular levels no auricular intrinsic deflections were seen.

Case 8 was that of a male, aged 69, suffering from coronary disease and hypertension, with congestive cardiac failure in varying degree for three years.

FIG. 2.—No auricular intrinsic deflections in the œsophageal cardiogram.

(A) Case 4: auricular fibrillation; subsequent normal rhythm.

(B) Case 7, (C) Case 8, (D) Case 9: auricular fibrillation; no return of normal rhythm.

Normal rhythm had been present two years before. Three weeks before admission he had a sudden increase in breathlessness and œdema, possibly indicating the onset of fibrillation, which was present on admission and remained. He was treated with digitalis, but was not given quinidine.

Standard lead cardiograms (Fig. 2 C), showed fibrillation with very small "f" waves having a maximum amplitude of less than 0.1 mv. Although œsophageal tracings were made from several auricular levels no auricular intrinsic deflections were seen; the auricular waves had a maximum amplitude of 0.2 mv. and a minimum of less than 0.1 mv.

Case 9 was that of a male, aged 60, suffering from hypertension. He was admitted with dyspnœa and œdema of the ankles of two weeks' duration. Auricular fibrillation was recorded several times during the first month when symptoms were severe, but normal rhythm appeared later as he improved under digitalis and salyrgan therapy. He remained well for three months after discharge, when dyspnœa and œdema suddenly returned; he was then readmitted. Although improvement again followed similar treatment, fibrillation was present throughout his three weeks in hospital. He received no quinidine.

Standard lead cardiograms (Fig. 2 D), taken during the second admission, showed fibrillation with very small "f" waves having a maximum amplitude of less than 0.1 mv. The auricular deflections in the œsophageal cardiograms, also taken at this time, were of moderate size having a maximum amplitude of 0.25 mv. and a minimum of less than 0.1 mv., but, although several auricular levels were examined, no auricular intrinsic deflections were recorded.

DISCUSSION

Since we have recorded auricular intrinsic deflections by the œsophageal lead in five out of nine cases of auricular fibrillation, it would appear that such deflections are more frequently present in œsophageal cardiograms in this arrhythmia than has been supposed. The present work fails to support Brown's contention (1936*b*) that they are to be found only in cases liable to spontaneous reversion to normal rhythm. They were found in one case of paroxysmal fibrillation (Case 1), and in two of fibrillation of recent onset, responding to quinidine (Cases 2 and 3), but also in one case of fibrillation and rheumatic heart disease with a six years' history of congestive failure (Case 5), and in one case of fibrillation and coronary disease with a year's history of congestive failure (Case 6); in the last two auricular fibrillation might be expected to remain established.

On the other hand, auricular intrinsic deflections were not recorded in one case of paroxysmal fibrillation (Case 4), in one case in which normal rhythm had been present five months before (Case 9), in another in which normal rhythm had been recorded two years before and in which fibrillation may have been present for only three weeks prior to admission (Case 8), and in a fourth in which fibrillation was known to have been present for two years before admission (Case 7).

The presence or absence of auricular intrinsic deflections in the œsophageal lead tracings of cases of fibrillation does not indicate the likelihood or otherwise of reversion to normal rhythm, for, of the four instances of such reversion, one

(Case 4) showed no intrinsic deflections although the fibrillation was paroxysmal, and, of the five cases in whom the arrhythmia persisted, two (Cases 5 and 6) did show intrinsic deflections.

The work of Garrey (1914, 1924) and of Brams and Katz (1931) has produced evidence that, in experimental animals, induced auricular or ventricular fibrillation is not necessarily associated with the development of a circus movement of the impulse or of control of the activation of the muscle by a central circulating mother wave. Brown (1936*b*) analysed two simultaneous tracings taken by œsophageal leads in a case of auricular fibrillation and came to the conclusion that no central circulating wave was present in the auricles; no intrinsic deflections were seen in these records. It seems reasonable to suppose, from the records published by Brown and by Deglaude and Laubry, as well as from our own, that in many cases auricular intrinsic deflections are not obtainable. In these, therefore, there is no sign that co-ordinated activation of large muscle masses takes place, at any rate in the posterior part of the left auricle, such as would be expected if a central mother circus wave existed.

On the other hand, our records show that there are cases of fibrillation in which many auricular intrinsic deflections can be recorded. In these simultaneous activation of large muscle masses lying close to the electrode occurs frequently. The repetition of the deflections suggests the repetition of the same electrical process and is compatible with a central circulating impulse.

Brown's study (1936*b*) of œsophageal cardiograms in auricular flutter provides valuable evidence in support of the theory of Lewis and his associates (1921, 1925) that this arrhythmia is due to the continual circulation of an impulse round a more or less constant path. Brown's published and our unpublished œsophageal lead curves of flutter and the direct lead curves of experimental flutter published by Lewis (1921) show a close resemblance in the form of the auricular complexes to those obtained by us in auricular fibrillation (see especially Case 3, Fig. 1 D); this is striking enough to suggest that a similar mechanism is involved. However, two differences are apparent, namely, that in fibrillation the auricular rate is higher than in flutter (in our cases from about 375 to 500 per minute), and that in fibrillation the intrinsic deflections occur in groups separated by irregular smaller waves, whereas in flutter there is a constant wave-form. Sometimes, as in Case 5, intrinsic deflections are sparse, occurring singly. If the similarity between the auricular complexes in flutter and the complexes in fibrillation with intrinsic deflections can be considered to show that a circus movement exists in the latter, it must be argued that the path of the circus varies. When intrinsic deflections are recorded the circus must have passed close to the electrode but when the auricular complexes are small, irregular, and slurred, the path of the circus must have moved away from the proximity of the electrode, and the muscle near the electrode must have been irregularly activated in small masses by offshoots some distance from the central circus. A gradual change from complexes showing intrinsic deflections to slurred complexes without intrinsic deflections, suggestive of a gradual movement of the path of the circus away from the proximity of the electrode, is shown in Fig. 1 F (Case 6).

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In the cases not showing intrinsic deflections long records were taken from various auricular levels. Since the œsophageal lead is capable of recording such deflections if long enough records are taken, it is probable that no mechanism involving the simultaneous activation of large muscle masses in the posterior wall of the left auricle or in the wall of the right auricle immediately medial to the entrance of the inferior vena cava was at work in these instances. In these cases the arrhythmia is unlikely to have been associated with a single circulating mother impulse.

Garrey (1924) stated that in experimental flutter and fibrillation there might be any degree of "circus movement" from a large-ring central mother circus in flutter to delirious fibrillation with many small ring-like circuits varying in location according to changes in the degree and position of local areas of block. The results of this study suggest that this hypothesis may be extended to human auricular fibrillation. At the one end of the scale are those in whose œsophageal cardiograms numerous intrinsic deflections occur, giving the tracings an appearance closely resembling those of auricular flutter. At the other end are those where the auricular deflections are small and irregular without any intrinsic waves.

SUMMARY

1. Nine cases of auricular fibrillation have been examined by means of the œsophageal lead; in four a return to normal rhythm was observed.

2. Auricular intrinsic deflections were obtained in five cases, one of paroxysmal fibrillation, two of recent fibrillation responding to quinidine, and two in which the arrhythmia remained established.

3. Auricular intrinsic deflections were not found in the remaining four cases, one of paroxysmal and three of established fibrillation.

4. No association was found between the presence of auricular intrinsic deflections in the œsophageal cardiograms of the patients studied and the likelihood of reversion to normal rhythm.

5. The mechanism of auricular fibrillation is discussed in the light of the curves obtained.

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