The Mechanism of Spontaneous Auricular Flutter and Fibrillation in Man

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ALTHOUGH auricular flutter and fibrillation have been studied for many years, most evidence obtained is relatively indirect and subject to various interpretations. Consequently, considerable controversy exists concerning the mechanism of these arrhythmias. Of the various investigative methods now available, only two provide definite evidence of the exact course of events occurring in the auricles. First, the auricular excitation wave may be traced by means of direct or semidirect leads from multiple sites on or adjacent to the auricular surface. Second, the auricular contraction wave may be clearly visualized in high-speed cinematographs. As described elsewhere, these two direct methods of observation have been extensively employed in experimental animals. More recently, the operations of auricular appendectomy and mitral commissurotomy have enabled us to observe directly the auricles of several patients during episodes of spontaneous auricular flutter or fibrillation. Further studies of spontaneous arrhythmias in man are now in progress and will be reported fully in subsequent communications.*

In the experimental animal, auricular flutter and fibrillation may be initiated by a variety of methods. The possibility exists that flutter or fibrillation produced by one method may differ in mechanism from examples of the same arrhythmia produced by an alternative method.† Moreover, it has been contended that experimentally produced flutter and fibrillation in animals may involve different mechanisms than their spontaneous counterparts in man. Since the experimental findings are of widespread interest only insofar as they elucidate clinical disturbances, the present discussion will be limited to (1) a description of electrocardiographic and cinematographic observations in patients with spontaneous flutter or fibrillation, and (2) a brief comparison between observations in man and experimental animals.

Clinically, auricular flutter and auricular fibrillation are closely associated. Both arrhythmias commonly accompany the same disease states. Moreover, conversion from flutter to fibrillation or from fibrillation to flutter often occurs either spontaneously or in response to drugs. Nevertheless, electrical and mechanical activity in the auricles of patients during spontaneous flutter has been found to differ radically from the activity characterizing spontaneous fibrillation. Observations on the two arrhythmias will therefore be presented separately.

Spontaneous Auricular Flutter in Man

The mechanism of spontaneous auricular flutter in patients was studied by the methods of simultaneous multiple esophageal lead electrocardiography, precordial and limb lead electrocardiography, and high-speed cinematography.

Electrocardiographic Findings.

At a distance of approximately 20 to 40 cm. from the lips, the human esophagus is in close association with both auricles. The esophageal method. No circus movement occurs when auricular flutter is produced by electrical stimulation, post-electrical stimulation or local application of aconitine.
region 30 to 37 cm. below the lips is at the midauricular level and is directly posterior to the interauricular septum. At the level of the extreme caudal end of the auricles, 37 to 40 cm. below the lips, the esophagus appears to be in more intimate contact with the right than with the left auricle. At levels 20 to 30 cm. from the lips, the esophagus is immediately posterior to the cephalic extremity of the left auricle. Although all portions of both auricles are not in direct contact with the esophagus, the distance separating these structures is so slight that electrical potentials in both auricles are recorded in esophageal leads from all auricular levels. Therefore, the course of the auricular impulse can be determined by analyzing the configuration of the depolarization waves in simultaneous esophageal leads. This has been accomplished in previous studies by other workers as well as in the present investigation of spontaneous auricular flutter in man.

Most patients with auricular flutter exhibit inverted depolarization waves in limb leads I, II and aVF. In these instances, representing the "common type" of flutter, esophageal leads from the caudal level of the auricles display a purely negative deflection wave, esophageal leads from the cephalic level of the auricles present a purely positive wave, and esophageal leads from the midauricular level contain a negative wave followed by a positive wave. In the uncommon type of auricular flutter, in which the depolarization wave is upright in limb leads I, II and aVF, negative depolarization waves are recorded in esophageal leads from the cephalic level and positive waves appear in esophageal leads from the caudal level; esophageal leads from the midauricular level exhibit biphasic deflections similar to those observed in the common type of flutter. Interpretation of these tracings is based upon the fundamental electrocardiographic principle that a negative deflection is inscribed as the cardiac impulse travels away from the recording electrode, while a positive deflection occurs when the impulse is traveling toward the electrode; a pure negative wave (called the intrinsicoid deflection), is recorded by an electrode at or near the site of origin of the impulse.

By applying the preceding principle, the esophageal lead electrocardiogram of spontaneous auricular flutter may be interpreted as follows: In the common type of flutter, the purely negative deflection recorded from the caudal level indicates that the impulse arises at and travels away from the caudal region of the auricles. Conversely, the positive deflection obtained in esophageal leads from the cephalic level establishes that the auricular impulse travels toward and terminates in the cephalic region. The biphasic deflection from the midauricular level shows that the impulse travels toward the middle of the auricle, passes beneath the recording electrode, then recedes from the electrode as it continues its journey to the cephalic extremity. Since electrodes in the esophagus record potentials in both auricles, the auricular impulse in these instances must originate from a caudal focus and travel in a caudocephalic direction through both auricles simultaneously. In the uncommon type of flutter, the impulse must arise in the cephalic region and travel through both auricles simultaneously in a cephalocaudal direction.

If a self-perpetuating circus movement were present in auricular flutter, as originally hypothesized by Lewis, identical deflections would be expected to occur in esophageal leads from all auricular levels since the circular path would be without beginning or end. Hence the above findings are inconsistent with Lewis' concept of circus movement. If a circus movement arose anew with each cardiac cycle, as suggested by certain workers, the deflections in esophageal leads from various auricular levels again would differ radically from those described above. In the common type of flutter, an impulse pursuing a circus pathway through both auricles would travel from the caudal region of one auricle to the cephalic extremity, where it would enter the opposite auricle and travel in a cephalocaudal direction on its return journey to the focus. Under such circumstances, electrodes in the esophagus at the caudal level of the auricles would register a negative deflection as the impulse traveled toward the cephalic region, followed by a positive deflection as the impulse returned to the caudal region. Similarly, esophageal leads
from the cephalic level of the auricles would exhibit a biphasic deflection, consisting of a positive component recorded while the impulse traveled up one auricle and a negative component recorded during the return journey. Finally, an electrode at the midauricular level would register a positive deflection as the impulse traveled from the focus to the middle of one auricle, a negative deflection as the impulse passed the electrode and continued to the cephalic extremity, and a second positive-negative phase as the impulse traversed the opposite auricle. Equally complex deflections would be recorded in the uncommon type of flutter if a circus movement was present. Thus, assuming that the fundamental principles of electrocardiography are valid, the configuration of the depolarization waves in esophageal leads from patients with spontaneous flutter is entirely incompatible with the occurrence of a circus movement traversing both auricles. Moreover, the above findings are equally inconsistent with the existence of a circus pathway around the septum, within only one auricle, or involving any anterolateral portion of one or both auricles. A comparison of the times of onset of the intrinsicid deflections in esophageal and precordial leads, as well as a calculation of the momentary auricular electrical axes in limb leads, likewise indicate that the excitation wave of spontaneous auricular flutter in man arises from an ectopic focus and pursues a linear rather than a circus pathway.

Certain patients with flutter arising from the cephalic region of the auricles exhibit depolarization (P') waves virtually identical with the normal P waves in limb and esophageal leads; that is, both the normal P and flutter P' deflections are upright in leads I, II, aV, and esophageal leads from midauricular levels, and inverted in esophageal leads from high auricular levels. It is generally acknowledged that the normal P wave is a graphic representation of the course of the auricular depolarization wave as it spreads radially outward from its site of origin. Since the P' wave of flutter sometimes is identical with the normal P wave, it is apparent that the flutter excitation wave in such instances likewise spreads radially from its site of origin rather than along a circus pathway.

Cinematographic Findings.

High-speed cinematographs of the exposed auricles have been recorded during mitral commissurotomy in a patient with spontaneous auricular flutter. Routine tracings from this patient exhibited inverted depolarization waves in limb leads II and III, indicating that the arrhythmia was of the common type arising in the caudal region. The auricular flutter rate was 240 beats per minute. Films were recorded at a camera speed of 100 frames per second and projected at a rate of 8 frames per second; hence the auricular contractions depicted on the screen were 13 times slower than the actual heart rate and occupied an interval of approximately 3.6 seconds per beat.

By pushing down on the pulmonary artery, it was possible to obtain films in which the motion of both auricular appendices was very clearly seen. If a circus movement had been present, either the left or the right appendix would have contracted before its fellow, depending on whether the movement were clockwise or counterclockwise. Indeed, assuming the distance between the two appendices constitutes one fourth of the hypothetic circus pathway, one appendix would have contracted about one second before the other. Actually, however, repeated examination of the cinematographs revealed that the two appendices contracted simultaneously. This phenomenon is entirely incompatible with the circus movement theory, but is consistent with the electrocardiographic observations described above. Cinematographically as well as electrocardiographically, therefore, it has been demonstrated that the excitation wave of spontaneous auricular flutter in man starts at an ectopic focus and spreads in a linear pathway through both auricles simultaneously.

Spontaneous Auricular Fibrillation in Man

Spontaneous auricular fibrillation in patients was studied by means of direct auricular lead electrocardiograms, esophageal and limb
lead oscillograms, and high-speed cinematographs.

Electrocardiographic Findings.

During operations on the mitral valve, direct auricular leads from four patients with spontaneous auricular fibrillation were recorded on the Sanborn Twin Beam Cardiograph. This photographic-writing instrument was operated at a paper speed of 75 mm. per second, yielding a much more accurate representation of the rapid electrical activity in the fibrillating auricle than is obtainable with standard electrocardiographic equipment. Examination of the tracings revealed slightly irregular waves varying from 0.2 to 0.4 mv. in amplitude and occurring at a frequency of 350 to 400 per minute. Some of the waves presented a sharp downstroke resembling an intrinsic deflection. No two waves were identical in shape or size. These deflections presumably correspond to the familiar "f" waves seen in limb leads of auricular fibrillation. In addition to the large waves, smaller waves of highly variable contour occurred at extremely rapid and irregular rates throughout the direct lead tracing, appearing as peaks or undulations along the baseline and on the upstroke and downstroke of the larger deflections.

Esophageal leads from 4 undigitalized and 20 digitalized patients with auricular fibrillation were recorded with the cathode ray oscillograph. Because of freedom from inertia, rapid recording speeds and virtually unlimited frequency response, the oscillograph proved even more satisfactory for purposes of the study than the photographic-writing cardiograph. In undigitalized subjects, esophageal leads from auricular levels revealed essentially the same phenomena as the direct leads described above. Large "f" waves, approximately 0.2 to 1.0 mv. in amplitude, were recorded at frequencies of about 350 to 600 per minute. Small waves, usually 0.1 mv. or less in amplitude, occurred at extremely irregular rates as high as several thousand per minute.

Simultaneous oscillograms from two electrodes placed 2.5 to 5 cm. apart in the esophagus revealed little or no synchronicity between the electrical activity in different parts of the fibrillating auricle. Esophageal leads from the digitalized patients exhibited deflections of much lower amplitude than records from undigitalized subjects but were otherwise essentially similar.

Cinematographic Findings.

High-speed cinematographs of the fibrillating auricles were recorded in four patients during mitral commissurotomy and in one patient during auricular appendectomy. Because of the presence of mitral stenosis, the left auricles of these patients were distended and often filled with clot; this factor, coupled with digitalization, limited the motion of the left auricle. However, in the patients undergoing commissurotomy, excellent cinematographs of the fibrillating right auricular appendix were obtained by pushing down the pulmonary artery.

When viewed in slow-motion pictures, the right auricular appendix was seen to contract at an irregular rate of 350 to 400 beats per second. Each contraction appeared as a more or less organized wave differing somewhat from the preceding wave in course, vigor, and rate of propagation. Careful examination of the films further revealed a second type of mechanical activity occurring simultaneously with the large contraction waves and consisting of rapid, heterorhythmic contractions and dilations of minute muscle segments throughout the appendix. These microscopic contractions were most readily discerned in the tip of the appendix where the fimbria twisted and turned first in one direction, then in another, in an entirely uncoordinated manner. Presumably the large contraction waves seen in the films correspond to the electrocardiographic "f" waves, while the microscopic contractions are mechanical counterparts of the small deflections recorded in direct leads.

Conclusions

From the preceding electrocardiographic, oscillographic and cinematographic observations, it may be concluded that spontaneous auricular fibrillation in the patient is a heterorhythmic disturbance composed of irregular large waves
which pursue a variable course over a sea of asynchronously contracting minute muscle segments. Neither the tracings nor the films reveal any evidence of a circus movement, or of an “isoelectric gap” or “daughter waves” such as those hypothesized by proponents of the circus movement theory.

**RELATIONSHIP BETWEEN CLINICAL AND EXPERIMENTAL ARRHYTHMIAS**

Except for differences attributable to pathology and medication, the preceding observations on spontaneous auricular flutter and fibrillation in man are remarkably similar to findings obtained during a four-year study of experimentally produced auricular arrhythmias in dogs. Direct leads and high-speed cinematographs from numerous experimental animals have shown that auricular flutter consists of rhythmic waves traveling from a focus at one end of the auricles to the opposite extremity through both auricles simultaneously, and that auricular fibrillation consists of heterorhythmic activity comparable to the large and small waves recorded from the fibrillating auricles of patients. The existence of a circus movement in the experimental instances studied has been conclusively ruled out. Although the operation of mitral commissurotomy has greatly facilitated investigation of the human auricle, much remains to be learned concerning the relationship between clinical and experimentally produced auricular arrhythmias. Nevertheless, it is noteworthy that findings in a large series of experimental animals are essentially similar to the results obtained by direct observation of spontaneous auricular flutter and fibrillation in man.
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