The Nature of Auricular Fibrillation and Flutter: A Symposium

By Herrman L. Blumgart, M.D.

Introduction

The idea of this symposium arose during an informal discussion at a meeting of the Editorial Board of Circulation. Much important evidence recently brought forward by the proponents of various theories of the auricular arrhythmias has been difficult to assess by those not intimately engaged in this field. It is believed that the present symposium by leading authorities clarifies some of the issues involved in the current debate. The admirable skill and lucidity with which the evidence for different concepts has been marshalled and ably summarized will, it is hoped, be of value in the clinical evaluation and further research of these arrhythmias.

The symposium will take the form of concise answers to four questions of fundamental importance by the participants. Each contributor will then elaborate upon his answers in a brief discussion.

Questions and Answers

Question 1. Has it been proved that auricular flutter and auricular fibrillation represent the same fundamental mechanism?

Hans H. Hecht, M.D. The Symposium implies that no agreement has been reached on the mechanism underlying the two disorders; consequently the question cannot be answered with factual assurance. There is much circumstantial evidence, accepted by everyone, that the two conditions are only quantitatively different and that whatever mechanism may be responsible, it forms the matrix for both. Experimentally, auricular fibrillation and auricular flutter can be induced by the same procedures and the identical clinical situation may be found with either, though flutter is less common and less easily elicited than fibrillation, it being apparently the less stable disorder of the two. Vagal stimulation by (a) shortening the refractory period and (b) increasing impulse transmission converts flutter to fibrillation. Quinidine and like substances by exerting the opposite effect, convert fibrillation to flutter. Intermediate forms or spontaneous changes from one to the other within seconds are often seen, particularly if suitable precordial or esophageal electrocardiographic explorations are employed, and an imperceptible transition can regularly be noted during digitalis or quinidine medication. Conversion of flutter to paroxysmal auricular tachycardia (usually with some degree of A-V block) is frequently possible, and, again, intermediate forms are often obtained during quinidine treatment. In man, this is less readily accomplished than the conversion of flutter to fibrillation, but the kinship between these two auricular disorders seems fairly obvious, at least in instances where these transitions can be observed.

Louis N. Katz, M.D., and Alfred Pick, M.D. The four questions which Dr. Herrman L. Blumgart has asked us to answer as a part of
the symposium on the nature of auricular flutter and fibrillation serve as the background of the personal opinion expressed by us in our formal presentation to this symposium, and our answer cannot be over-simplified. The reader, therefore, must read our personal opinion to see how we would have answered these four questions. However, if we must summarize our viewpoints, our answer to the first question would be as follows:

It has not been proved that the fundamental mechanism of auricular flutter and auricular fibrillation represents the same fundamental process, but the evidence for this is highly suggestive.

Myron Prinzmetal, M.D. Direct electrocardiographic and cinematographic evidence obtained from numerous experimental animals has established that both auricular flutter and auricular fibrillation occur when an ectopic focus on the auricles discharges at rapid rates. Moreover, spontaneous or drug-induced conversions between the two arrhythmias are commonly seen clinically as well as experimentally. These observations indicate that auricular flutter and auricular fibrillation represent the same fundamental disturbance, namely, the rapid discharge of impulses from an ectopic focus, but differ primarily in the rate of auricular activity. However, the mechanical and electrical activity observed in the auricles during flutter and fibrillation is radically different, in some instances presumably because the auricular musculature is unable to maintain organized conduction when the auricular rate exceeds the fibrillation threshold.

Arturo Rosenblueth, M.D. No, but it is very likely that they have the same mechanism.

Question 2. Has the mechanism of auricular flutter and auricular fibrillation been established in the animal?

Hans H. Hecht, M.D. The answer here is no. A circus excitation and circus contraction have been unequivocally demonstrated in lower animals under appropriate conditions, as a characteristic pattern of response of cardiac tissue of certain mass and form. In dogs, a repetitive ectopic focus or an area subjected to continuous stimulation has given rise to mechanical and electrical records that at least closely resemble the spontaneous auricular flutter and auricular fibrillation in man. Older experiments (Garrey) have demonstrated that isolating the focus from the remainder of auricular tissue did not influence or interrupt the irregularity, and this has been used in support of the circus excitation concept. Newer observations (Scherf, Prinzmetal), using a similar technic but different stimuli, have shown that the irregularity is interrupted if the focus is severed from the remainder of the auricles, and this is claimed as evidence in favor of a continuously firing single focus. Rosenblueth has recently published a technic of inducing and maintaining a flutter-like disorder in dogs that indirectly supports the concept of circus contractions. This technic has been employed in various laboratories and his observations have been repeatedly confirmed.

Louis N. Katz, M.D., and Alfred Pick, M.D. No. The mechanism of auricular flutter and auricular fibrillation has not been established, but the possibilities in the animal have been narrowed, and there is reason to believe that auricular flutter may include the primary and secondary types of mechanism as outlined in our brief paper which follows.

Myron Prinzmetal, M.D. Auricular flutter produced by local application of aconitine or as an after-effect of electrical stimulation has been demonstrated to consist of rhythmic impulses arising at rapid rates from an ectopic focus and traveling outward in all available directions simultaneously. Auricular fibrillation initiated by similar methods consists of heterorhythmic waves of variable strength, frequency, and velocity occurring simultaneously and superimposed on one another. It should be pointed out that certain workers experienced in the use of Rosenblueth’s crush method for producing arrhythmias believe that flutter initiated by this technic involves a circus movement.

Arturo Rosenblueth, M.D. Yes. The mechanism is circus-movement.
Question 3. Has the mechanism of auricular flutter and auricular fibrillation been established in man?

Hans H. Hecht, M.D. The answer again is no. No conclusive observations have been presented to date in support of one or the other of the theories in spite of almost four decades of heated assertions.

Louis N. Katz, M.D., and Alfred Pick, M.D. No. The mechanism of auricular flutter and fibrillation has not been established in man, but the analogy to the animal suggests the same conclusion we have expressed in our answer to question 2.

Myron Prinzmetal, M.D. By means of esophageal and limb-lead electrocardiography as well as high-speed cinematography, auricular flutter and auricular fibrillation in man have been shown to be essentially similar in mechanism to the experimental forms of these arrhythmias produced in dogs by chemical or electrical stimulation.

Arturo Rosenblueth, M.D. No.

Question 4. In the light of present knowledge, which mechanism do you believe to be responsible for auricular flutter and/or auricular fibrillation in man?

Hans H. Hecht, M.D. The apparent similarity of human records of the auricular dysrhythmias under discussion to those experimentally induced does not constitute incontestable proof that they are identical. Even if this were assumed, the differences presently existing in interpreting the basic nature of the disorder as seen in animals only multiply when attempts are made to apply the observations to the clinical disorder in man. The facts that high intraluminal pressures, anoxia, vagal stimulation and other influences induce the irregularities or that a certain spread of auricular excitation may be observed grossly does not favor one concept over the other; changes in the refractory period may predictably influence the characteristics of the disorders irrespective of their presumed mechanism. No common ground can be found which would reconcile fully the views supported by various investigators, nor does it seem possible to disregard one set of experiments in favor of another. It is possible that multiple mechanisms either singly or together may cause the admittedly heterogeneous experimental and clinical picture of auricular flutter and auricular fibrillation. In fact, the analysis and scrutiny of known observations makes it likely that this is so. Large circular paths as well as small areas responding in chain-like fashion with continuous re-entry of impulses either with or without persistent ectopic stimulus, or a stimulus without re-entry phenomena could, under appropriate circumstances, disrupt the orderly manner of auricular excitation and result in extrasystolic disorders, paroxysmal tachycardia, auricular flutter and auricular fibrillation.

Louis N. Katz, M.D., and Alfred Pick, M.D. As regards the question of choosing a mechanism to account for flutter and/or fibrillation, this cannot be summarized in a few words. The answer must be sought in our complete presentation, which follows. Any attempt to abbreviate carries with it the hazard of being consciously or unconsciously misinterpreted by vehement protagonists of contrary views.

Myron Prinzmetal, M.D. With the development of new technics of cardiac surgery, it has become possible to study the spontaneous arrhythmias in man by direct methods such as high-speed cinematography and direct lead electrocardiography. These methods have already yielded convincing evidence that the clinical forms of auricular flutter and auricular fibrillation, like their experimentally produced counterparts, do not involve a circus movement. In flutter, the excitation wave travels away from the focus through both auricles simultaneously; in fibrillation, the auricular musculature exhibits heterorhythmic activity different from that observed in any other auricular rhythm.

Arturo Rosenblueth, M.D. I believe that both auricular flutter and auricular fibrillation in man are due to circus movement of impulses around appropriate obstacles.
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