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Auricular fibrillation including a survey of seventy four cases

Walter P. Senter
University of Nebraska Medical Center

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AURICULAR FIBRILLATION

including

A Survey of Seventy-four Cases

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W. P. Senter

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Concerning Theses

The origin of theses for degrees dates back to very ancient times, having grown out of disputations carried on publicly between two persons of opposing opinions. This intellectual exercise, which is almost coeval with the human race, appears to have been formally initiated by St. Bonaventure, Superior of the Franciscan Order of Monks, in the thirteenth century, in order to control the instruction given by the teachers and to test the progress made by the students; in 1266 St. Bonaventure himself sustained a thesis at Paris.

From 1323 onwards these disputations, for though called theses they did not exactly correspond to those we are now familiar with, formed part of the obligations for the degree of Magister Artium at the Sorbonne, Paris. The author of the article "These", in Larousse's Dictionnaire universel (1876, xv, 107) remarks that "thèses are still employed in the Universities, but are less elaborate and less important, and a great number of those for the Doctorate of Medicine are unfortunately only a vain formality." These were first written in the language of learned men, namely, Latin, and this cus-

tom was long preserved. Sir George Humphry, one of the most impressive teachers I have ever had, but not a great classic, was obliged to write his thesis for the M.B. Cambridge 1842 in Latin. Apparently the rule about reading theses in Latin was relaxed in 1858, for Dr. J. N. Keynes, the Registrary of the University of Cambridge, has kindly informed me that the regulation passed in May of that year is to the effect that "the candidate shall read a thesis composed in English by himself," etc. Sir George proceeded to the M.D. degree in 1859.

The objects of a thesis are (a) to show how far the mind has been trained; (b) to stimulate the mind and to make the candidate think, so that he may be qualified not only to deal with problems but to report logically and intelligibly on what he has observed; (c) although it is not a necessary part of every medical man's life to write papers on medical subjects, the elaboration of a thesis, if taken seriously, is a valuable though brief education in this method.[1]

"I state with firm conviction that no one can appreciate the essentials in the recognition of heart disease and heart failure until he has made himself familiar with the varied phases of auricular fibrillation."

--Mackenzie

Preface

The usual undergraduate thesis consists of material from several sources scrambled together to make a readable whole. In order to instill a semblance of originality into this paper it is intended that it embody, in part, information gathered from the study of case-records.

In the beginning it was expected that at least one-hundred cases might be studied. However, when readmittances were classified under their respective original admittances the number of available cases dwindled to seventy-four. The seventy-four cases herein surveyed were patients in the University Hospital during the period 1917 to 1930.

W. P. S.

AURICULAR FIBRILLATION

Definition

Auricular fibrillation is the term most commonly applied at the present time to a cardiac arrhythmia characterized clinically by a variation in the timing and magnitude of successive ventricular contractions, and electrocardiographically by a disappearance of the P wave, irregular sequence of the ventricular complexes, and the presence of small irregular oscillations between the ventricular complexes.

The term itself has many synonyms. Hence we read of the delerium cordis and mitral pulse of the older writers; the perpetual irregularity of Hering; the complete arrhythmia of Josue; the nodal rhythm and paralysis of the auricle of Mackenzie; and finally, the auricular hypertachyrhythmia of Geraudel.

Historical

Several hundred years ago clinical observers were dimly conscious of a peculiar condition of the heart which produced ill-health in certain people.

The description by Withering, of the effect of digitalis, shows that he recognized a form of heart failure which was peculiarly susceptible to the drug. After his time, when physicians recognized mitral stenosis, they came to associate a peculiar pulse which frequently was present in this condition, to which they gave the name "mitral pulse".

Quoting from Bramwell, "Diseases of the Heart" 1884: There has been a good deal of debate with regard to the rhythm of the heart in mitral stenosis. The majority of observers are, however agreed--and with their opinion I entirely concur--that the pulse is irregular. Dr. Sansom--than whom no better authority could be quoted says, "We may conclude, therefore, that a pulse tracing which shows irregularity in the diastolic periods, sometimes missed pulsations, and as described by Dr. B. Foster, the occasional appearance of a small abortive pulsation in the line of descent, is a very strong evidence of the existence of mitral stenosis". Dr. Mahomed who was, I believe, the first to describe this peculiar rhythm as characteristic of mitral stenosis, has since somewhat modified his views. He says, "Although it is very commonly present in this disease, perhaps more commonly than in any other valv-

ular lesion, nevertheless it is not so much a sign of stenosis of the valves as of a dilatation of the ventricle. It is true, that in the typical cases of mitral stenosis the ventricle is not dilated, but I am unable to say whether this irregularity only occurs in cases in which dilatation exists."

Mackenzie, in his experiments on the venous pulse discovered about thirty years ago that in certain patients all evidences of auricular activity suddenly disappeared, and to this observation we may date the inception of systematic study of the condition now termed auricular fibrillation.

Nature of Auricular Fibrillation

Hypothesis of Paralysis of the Auricle--This, the earliest explanation of auricular fibrillation, was offered by Mackenzie. With the disappearance of the auricular wave a, in tracings of the venous pulse, he assumed that the auricle ceased to beat and that the ventricle started on a rhythm of its own. This hypothesis was soon abandoned, due to the fact that at autopsy the auricles were not found to be atrophic, but rather hypertrophic, and it was difficult to conceive how a paralyzed auricle

could be hypertrophied.

Hypothesis of Nodal Rhythm and Sino-auricular Block--Mackenzie in 1904 modified his views, and attributed the absence of evidence of the auricular contraction in tracings of the venous pulse to displacement of the motor stimulus from the sino-auricular node to that portion of the primitive bundle that corresponds to Tawara's node. He reasoned that since this node is situated midway between the auricle and the ventricle, the excitation wave must necessarily take the same time to reach the two chambers and simultaneous contraction of auricles and ventricles result. In other words the tracing of the auricular contraction was lost in the tracing of the simultaneous ventricular contraction. According to Mackenzie, the displacement of the motor stimulus was due to a heightened excitability of Tawara's node: according to Wenckebach, to an interruption in the means of communication between the sinus and the auricle, a sino-auricular block.

This hypothesis must be rejected because it does not account for the essential feature, the irregularity of the beats which, together with the disappearance of wave a, characterizes the condition. Moreover, in all cases in which undoubted

nodal rhythm has been produced experimentally, the beats have been coordinated and regular. Wenckebach's explanation is no more acceptable for the same reason. In fact, Hewlett has shown that in experimental sino-auricular block both the auricular contractions and the normal rhythm are preserved.

Hypothesis of Fibrillation--In 1849, Ludwig and Hoffa noticed that on exciting any portion of the dog's heart with the faradic current, the rhythmic pulsations gave place to incoordinated fibrillary tremors. This phenomenon was rediscovered later by Ludwig, then by Vulpian, MacWilliam and Winterberg in the ventricles and by Philips and Fredericq in the auricles. These latter investigators showed that the fibrillary contractions of the auricles excited by weak currents were accompanied by corresponding changes in the rhythm of the ventricles, which returned to normal as soon as the auricles had ceased to fibrillate. Subsequently it was observed that the effects of mechanical irritation of the vagus nerve were identical with those produced by the electric current. The same is true of intravenous injections of certain poisons, such as digitalis, aconitin, nicotin, and the alkaline salts. Furthermore, as early as

1899, Cushny had observed that the tracings taken in these experiments showed strong resemblance to those from patients suffering from auricular fibrillation. In 1907, with Edmunds, he returned to the subject and suggested that the arrhythmia was caused by auricular fibrillation. In 1909, Magnus-Alsleben expressed the same opinion. Final proof was given by Einthoven, Kraus and Nicolai, and Hering by means of electrocardiography. These authors showed that the electric curves of a fibrillating dog heart could be superimposed exactly on those of patients suffering from the arrhythmia. These conclusions have been confirmed by Winterberg and Rothberger[9], Jolly and Ritchie[11], and Lewis[10]. Accepting the hypothesis of fibrillation, several theories are advanced as to whence proceeds the excitation.

Theory of Monotopia--This theory, defended by Rothberger and Winterberg, assumes that the stimulation proceeds from a single center. This is not the sino-auricular node, but a heterotopic center which emits a rapid series of excitations. The refractory phase of the auricular tissue would then be extremely short. It would result from these special conditions that the portion of the myocardium close to the center of excitation would cease

to be refractory and again become irritable, while the portion of myocardium more remote from this center would be in the phase of contraction. A new excitation would then act on the near portion while at the same time the preceding excitation was acting on the distant portion. Two or more waves of excitation starting from the same center would follow one another then in their auricular course.

Theory of Polytopia--This theory, first presented by Englemann, accepted and then abandoned by Lewis, has again been defended by Kisch. A stimulus of a nature not specified originates at numerous points of a hyperexcitable myocardium and engenders contractions which, joining each other or interfering, give rise to the irregular events of fibrillation. One objection to this theory is the fact of experience that a normal rhythm may abruptly follow the phase of fibrillation. The sino-auricular node would then have resumed control. It is inexplicable that the accessory centers should all be arrested at the same instant.

Theory of Fractional Contraction--DeBoer proposes that excitation is supplied by a single center, which may be the sino-auricular node, and concludes that when the myocardium is well nourished, its

phase of contraction occurs everywhere at the same time, and it is the same for the refractory phase, but when the myocardium is poorly nourished its different portions are not all in the same phase at a given moment. Some parts are in contraction, others in the refractory phase, others again in a non-refractory phase. When an excitation is launched through this mosaic of myocardial territories in different phases, its course will be irregular and it will cause a series of partial contractions which succeed one another in jerky fashion, whence the tracing of fibrillation.

Theory of Circus Contraction--Lewis supports the theory of "circus contraction"[6,7] or "circus movement". It rests on an experiment made on a muscular ring taken from a jelly-fish[14]. If this muscular ring is irritated at a definite point, a wave of contraction starts to the right and left of the region affected. The two waves uniting at the opposite side of the ring annul each other and the contraction is arrested. But if a circumscribed part of the ring is lightly compressed by a forceps and a point, situated at the right, for example, of the region compressed is immediately stimulated, there is only one wave of contraction, that going

to the right. The wave going to the left is blocked by the part compressed. If then, the compression is loosened before the wave of contraction in its course around the ring has reached that region, the blockade is raised, and the wave of contraction passes the segment recently blockaded and extends beyond the point from which it started, the refractory period of which, following the first contraction, having just ceased. The wave of contraction always finding in front of it a portion of the ring which has just emerged from its refractory period, follows it indefinitely. In practice, as a consequence of a single stimulus, a wave of contraction has thus circulated for hours in a muscular ring.

An analogous result has been obtained with a ring from the heart of a tortoise. From his experiments on the dog, Lewis has concluded that the course followed by the circus contraction surrounds the mouth of one of the vena cavae and is sinuous and so sends out sinuous centrifugal excitations. He explains flutter in the same way although here he believes the path of the circus movement to be a regular one and probably includes the mouths of both vena cavae.

It may be objected to Lewis's theory of the circus contraction that it is difficult to understand

the persistence of this circular course of the wave of contraction, for it is admitted that it may penetrate equally into other muscular territories. One cannot, then comprehend how the circular wave alone finds expression in the electrocardiogram while the centrifugal radiating waves do not manifest themselves.

At the present moment this, of all theories of fibrillation seems to be the most widely accepted.

Hypothesis of Geraudel --Geraudel has recently called attention to a serious omission, that no account has been taken of the incidental role of the circulatory system in the mechanism of the heart beat and brings forth a new theory of such mechanism and incidentally a new explanation of auricular fibrillation.

The classical theory of the cardiac mechanism rests essentially on the idea that the auricle is closely connected with the ventricle by a special bundle, the bundle of His, whence the coordination of their movements.

This coordination is lacking oftentimes: Even in the normal state, where a certain play may exist between the auricle and ventricle; still more when the heart is ailing, the independence of the auricular and ventricular rhythms being then at times

so modified that we can no longer speak of coordination, however loose we may imagine it to be.

The bundle of His has the same structure as the sino-auricular node to which is attributed a very different function, namely, that of a motor center. Contrary to this opinion, Geraudel attributes the same function to these two similar formations which he calls the cardionectors, the sino-auricular node, or atrionector, being to the auricle what the bundle of His, or ventriculonector, is to the ventricle.

In experiments with direct electrocardiography, Athanasiu registered certain electric undulations which he thinks indicate a nervous influence. The rhythm of these waves is about six-hundred per minute. This is precisely the maximal rhythm at which the auricle works in auricular fibrillation. To Geraudel it seems probable then, that each cardionector struck by the waves with this rate, permits or not, the passage of this permanent undulatory current and at a fixed rhythm. It acts like a connector; whence the name cardionector.

Thus the atrionector and the auricle on the one hand, and the ventriculonector and the ventricle on the other, constitute two parts which act independently of each other. This conception is further confirmed by the distribution of the vessels of the

heart. He has, in fact, shown that each cardionector has its own circulation with one terminal artery for the atrionector and another for the ventriculonector.

It is therefore logical to suppose that a circulatory disturbance may affect only one of the cardionectors, or both, although differently, as a result of which, the functioning of the auricle and that of the ventricle are seen not to be in partnership. So long as the circulatory equilibrium between the two cardionectors is maintained, the rhythms of the auricle and ventricle remain equal. The independence of the two cavities is then masked and auricle and ventricle seem to be intimately associated with each other. This is the case with the normal heart. But when circulatory disturbance supervenes, however slight it may be, the equilibrium will be broken, and the auricle and ventricle will beat each at its own rate; their independence will thus be revealed and their assumed coordination will cease, The mechanism of the heart will become abnormal.

He believes, then, that it is its own circulation which is the chief regulatory mechanism of the heart. The anomalies are due less to the condition of the heart itself, than to that of its blood vessels.

Auricular fibrillation then, originates always from the overactivity of the atrionector which rules the functioning of the auricle. This overactivity may result from nervous, humoral, or toxic factors. When these factors act on the atrionector alone or more upon it than on the ventriculonector there will result a discordant auricular hypertachyrhythmia, as Geraudel chooses to call the condition. If the factors act equally, the ventricular rate will equal the auricular.

The overactivity of the cardionectors, especially that of the atrionector, which brings about the auricular hypertachyrhythmia, seems to be more frequently closely associated with an increased blood circulation in this atrionector. The hyperemia may be active through increase of the arterial circulation, but it is probable that there is more frequently a passive hyperemia, due to some obstruction to the venous outflow.

The auricular acceleration may then in its turn increase the obstruction to the intra-auricular venous circulation and so increase the passive hyperemia not alone of the atrionector but of the ventriculonector as well. This in turn, occasions a more rapid ventricular contraction which further increases

the hyperemia and so contributes to the acceleration of rhythm. In this way is created a sort of vicious circle manifested by a crisis of double tachyrhythmia--auricular and ventricular. The paroxysm ceases only when the vicious circle is broken by the occurrence of a ventricular pause causing anemia of the cardionectors.

Ventricular arrhythmia, also seems to cause disturbances of the venous circulation of the cardionectors. These troubles increase with the acceleration of the auricular rate. The right auricle tends to become distended and its contractions are but little or not at all effective--all conditions eminently favorable to hinder the emptying of the veins of the cardionectors and to prolong unduly their contact with a modified fluid medium.

The condition is much the same in patients having a mitral lesion and consecutive dilatation of the right heart, especially of the right auricle, where we find fibrillation with extreme frequency.

Arrhythmia of the ventricle disappears or is lessened under two conditions--either when the beat is too rapid or when it is too slow. This is, Geraudel believes, because in the first case the ventriculonector, already in a state of active arterial

hyperemia, is less susceptible to the variations of venous hyperemia. In the second case the dominant element which influences the functioning of the ventriculonector is its arterial ischemia which tends to compensate the venous stasis.

The independence of the two cardionectors and their circulation accounts for all observable combinations, in which different modalities of functioning of the auricle and ventricle--acceleration, retardation, regularity, irregularity--are associated.

Geraudel also disbelieves fibrillation per se; as follows: It is possible that the auricle may fibrillate, that is to say that its wall may be the seat in man of parcellate or fibrillary contraction. This has been observed experimentally in animals when the heart is anemic or when the muscle is electrically excited. But the facts classed under the name of auricular fibrillation and which correspond to the tracings considered as characteristic of this anomaly of auricular functioning seem, in the absence of proof to the contrary, to originate from a total contraction of the auricular walls, more rapid and less regular than in flutter, but of identical nature; not from parcellate fibrillation. It is difficult to range in the same category the

transitory phenomena observed in the experimental animal, phenomena which are often preagonic, and the permanent phenomena seen in man which may, barring accident, persist for years.

Geraudel's theory has been presented in some detail mainly because it is new and possibly not well-known in this country. Needless to say, his work will require confirmation.

Etiology

Although the exact etiology of auricular fibrillation is not known, it is generally admitted that almost all cases of auricular fibrillation can be grouped under three heads. First, cases which present a thyroid history; second, cases presenting a rheumatic history; and third, cases lacking in a history of thyroidism or rheumatism, and for want of a better name classified as fibrotic.

Various data will now be presented under the three group headings.

Thyroid Group

In our series there were eight patients with a

thyroid history or approximately ten percent of the total cases of auricular fibrillation studied. Three were cases of exophthalmic goitre and five were toxic adenomata. Willius and Boothby[8] state that auricular fibrillation occurs with equal frequency in patients with exophthalmic goitre and with toxic adenomata, and is more apt to occur in cases of long duration than in cases of acute hyperthyroidism. Osler[23] finds the condition to be more frequent in the toxic adenoma cases, which is in agreement with our findings.

All of the patients in this group were female whites. The age range in the exophthalmic cases was 28 to 54, with an average of 40. The patients with toxic adenomata were older, the range being 45 to 66, and the average 54.5.

One case of toxic adenoma was complicated with a rheumatic history.

Rheumatic Group

Thirty-seven patients or 50% presented a definite history of acute rheumatic fever or chorea. The relative frequency of cases with a rheumatic history necessarily varies as the incidence of rheu-

matic fever in the community. Thus, Lewis[5] reports 66% in England, and in Boston Levine found a clear rheumatic etiology in 33%. On the Pacific coast auricular fibrillation of rheumatic origin appears to be even less frequent.[4]

The sexes in this group were almost equally divided, 19 male, and 18 female. Lewis states that in his series of 100 subjects 53 cases occurred in males and 47 in females. Apparently, then males are somewhat more vulnerable.

All of the negro patients, five, fell in this group.

The age range was from 19 to 67, with an average of 48. As might be expected, the youngest patient with auricular fibrillation belonged to this group.

The age-frequency may be seen in Table 1.

These figures are at variance with those of Lewis[5]. In his series of 91 cases he found the incidence to be heaviest between the twentieth and thirtieth years, and almost as heavy in the fourth and fifth decade. Perhaps this is due to a difference in the severity of rheumatic fever in this locality and England. Then too, a possible difference in racial resistance must be

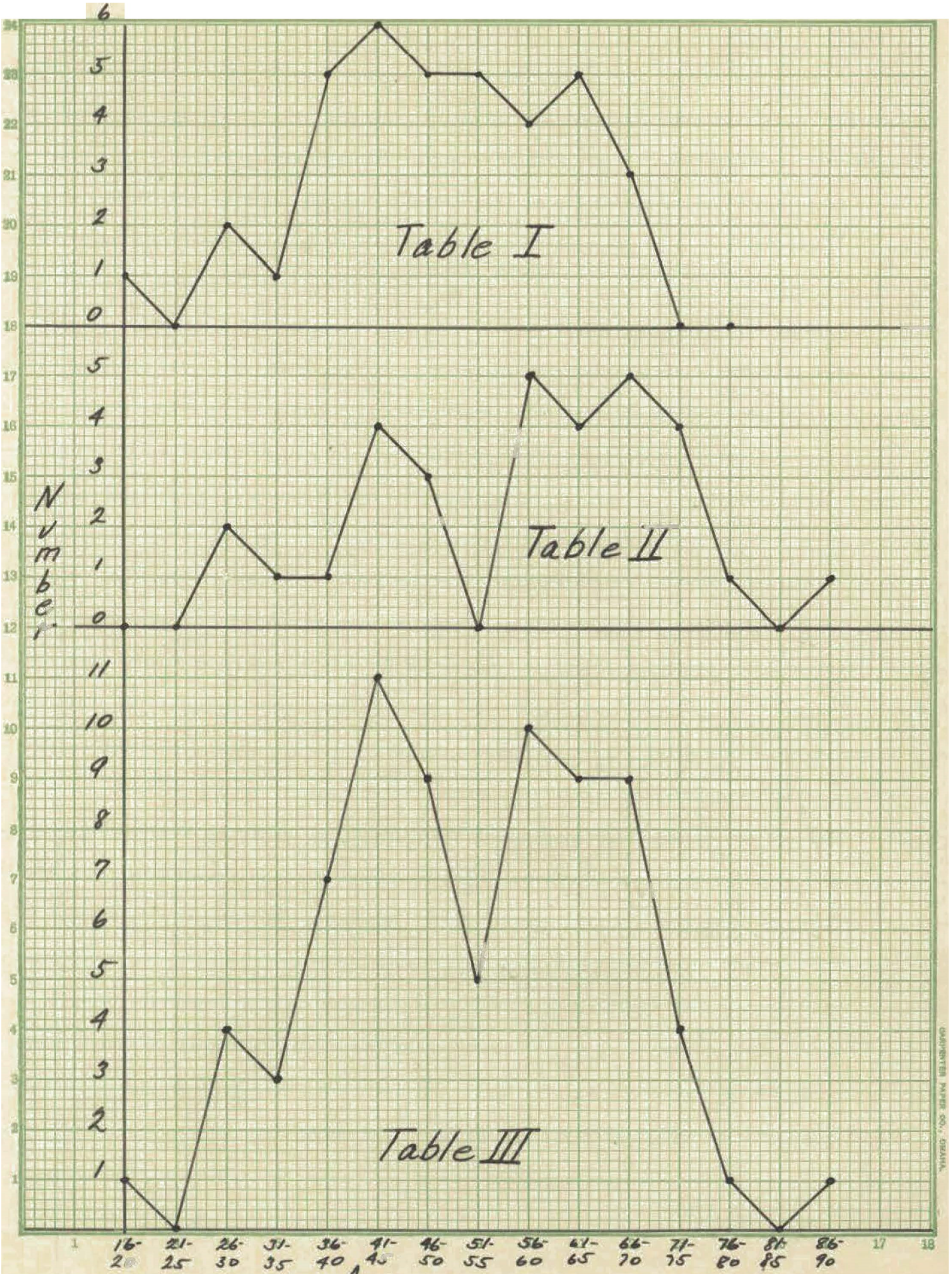
Table I
Table II
Table III
Number
16-20
21-25
26-30
31-35
36-40
41-45
46-50
51-55
56-60
61-65
66-70
71-75
76-80
81-85
86-90

thought of. At any rate, in this locality, auricular fibrillation seems to make its appearance some ten years later than in England.

Fibrotic Group

The fibrotic group consisted of 31 cases or 42% of the total. Here the sex incidence was well marked, 19 of the patients being male and only 12 female. All were of the white race. The ages of the patients ranged from 28 to 86 with an average age of 59. Fibrillation in this group appears at a later age generally speaking, than in the other two groups. The age-incidence is shown in Table 2.

Lewis[5] finds the highest incidence in the sixth and seventh decades which is in agreement with our findings.

Fibrillation in General

From a study of the foregoing groups certain facts may be stated concerning fibrillation in general.

Incidence--During the period 1917 to 1930 a total of 38,342 patients were admitted to the University Hospital. Of this number there were 74 cases of auricular fibrillation making the incid-

ence of this condition approximately .2%. In this connection it is interesting to note that the average period of hospitalization per patient was 44 days.

Sex--In this series of cases the condition was rather more frequent in the male sex, 38 cases or 54%. This in the main is in agreement with Lewis, who in a series of 189 subjects found 114 or 60% of the cases in males.[5]

Race--Ninety-three percent of the cases occurred in patients of the white race. This figure cannot be properly evaluated inasmuch as the proportion of whites to negroes in the total hospital admissions is not forthcoming.

Age--The youngest case of auricular fibrillation was aged nineteen years and the oldest eighty-six--average age 53. Table 3 shows the age-incidence of all cases. Lewis observed age limits of 5 to 84 and states that the condition is extremely rare before the age of 17.[5]

Pathology

The pathology of auricular fibrillation has been and is still the subject of much discussion.

Merklen, Rabe, Dehio, and Radesewski have described changes in the wall of the right auricle. Schoenberg noted lesions of the connections of the sinus node with auricles, that is, of the connecting bundle described by Wenckebach, the existance of which is not generally accepted. Edinger has found myocarditis at the mouths of the venae cavae, and Draper and Lenoble report lesions of the sinus node. Romis reported a case in which there was a sclerosis of the central part of the sinus node, and Koch described a dissociation of the auricular fibers by leucocytic infiltration. Stienon examined 20 hearts from subjects who presented auricular fibrillation, and in all of them he found one or the other of the lesions enumerated above. However, he states that they are just as pronounced in all hearts of stasis which have never shown auricular fibrillation.

Aschoff, Sternberg, and Monckeberg found no changes in the microscopic anatomy of the heart capable of explaining the disordered rhythm. Cohn was unable to find any lesion of the auriculoventricular node, of the trunk or the branches of the primitive bundle in horses that presented auricular fibrillation, which is very frequent in these animals.

Ribierre and Giroux in 1922 reported a case concerning a subject who had received a bullet wound in the left chest and who was seized immediately with cyanosis, dyspnea, dilatation of the heart, and auricular fibrillation. On autopsy a hematoma was found infiltrating and dissociating the fibers of the right auricle near the mouth of the superior vena cava.

The preceding observation is interesting in light of the conception of Geraudel. He produces no anatomic proof, but states that in both fibrillation and flutter ther exists a hyperemia of the cardionectors. For the opposite condition, bradycardia, definite organic causes of cardionector ischemia--partial occlusion of the arteries of the cardionectors--are shown, and he apparently reasons backward from these observations.

At autopsy, the auricles which have been the seat of fibrillation are often markedly dilated. Possibly this dilatation is a factor in producing the fibrillation, as may well happen in mitral stenosis. On the other hand, fibrillation itself favors dilatation, and in some instances the auricles are not dilated at all.[4]

In our series twelve cases came to autopsy.

The findings in the hearts of these patients were practically all gross, such as coronary sclerosis, fibrous infarction, myocardial degeneration, dilatation, hypertrophy, thickened valves, etc. In no case were the findings peculiar to auricular fibrillation.

There apparently is not, therefore, a specific histological syndrome in auricular fibrillation[17] known at the present time. However, as Geraudel points out, the finer circulation of the heart will bear much more painstaking and complete microscopic examination.

Symptoms

The entrance complaints of the hospital patients with auricular fibrillation were mainly those of circulatory inadequacy: Dyspnea, orthopnea, precordial pain, edema, headache, weakness, insomnia, palpitation, cough, epigastric distress, loss of appetite, pleuritic pain, tachycardia, liver pain, eructations, epistaxis, ascites, and pleural effusion. There were two cases of acute rheumatic fever who in addition complained of tender joints and fever. The thyroid cases also complained of

nervousness and loss of weight. Lewis[5] states that patients with auricular fibrillation are more prone to exhibit symptoms of over-taxation of the heart than those with similar valve lesions and a like degree of cardiac dilatation. This is due to the fact that the disordered action of the ventricle puts an added strain on the myocardium.

In this series the patients dated the onset of noticeable cardiac distress from several days to several years before hospitalization. In the majority of cases the time of onset was placed at less than one year.

The mode of onset varies considerably in different patients. As a rule in the course of making an effort such as going rapidly up a hill or running, or lifting a weight, the patient becomes conscious of his heart running away, or he suddenly becomes very breathless. Mackenzie[3] states that every case of heart failure with respiratory distress from what is called heart-strain which followed some extra effort could be attributed to the onset of auricular fibrillation or its allied condition of flutter.

In some patients there is not this distinctive onset but a history of a gradual increase of breath-

lessness. In these cases the fibrillation does not at first cause appreciable distress, but finally the inefficient action of the heart makes itself felt as a limitation of response to effort.

In still other patients attacks of fibrillation occur paroxysmally, manifesting themselves in breathlessness and limitation of activity. The arrhythmia may continue for more than twenty-four hours, then it decreases gradually and at a certain moment the patient's pulse may be found to have become regular. In some cases the termination of the paroxysms is abrupt like the onset. The paroxysmal type shows a tendency toward the establishment of a permanent fibrillation. In some patients the paroxysmal phase is very long--33 years has been reported by Heita. The coalescence of the paroxysms usually takes place by progressive lengthening of the paroxysms and shortening of the intervals between them.

Rarely the condition is discovered in patients in the course of a routine physical examination, the patient being unaware of the condition and suffering no distress from it.

Diagnosis

Auricular fibrillation is in most cases comparatively easy to diagnose. In many patients one suspects the presence of auricular fibrillation at the first glance. The patient usually presents a dusky face with blue lips and ears, and labored respiration on moderate effort, as a result of diminution of the output of blood from the heart due to the many....small, imperfect, and ineffective beats of the ventricle.

The pulse is irregular in time and intensity, that is, completely so, and there is a discrepancy between the rate at the apex and at the wrist. This condition is known as a pulse deficit and is the result of ventricular contractions of insufficient intensity to reach the wrist.

The ventricular rate is usually rapid, and the statement is made that an irregularly beating heart with a rate of more than 120 beats per minute is almost certainly the seat of fibrillation. In a slowly fibrillating heart a mild amount of exercise will increase the rate and make the irregularity more evident clinically.

In the absence of murmurs or in the presence

of systolic murmurs the irregularity is the distinctive feature found on auscultation. When mitral stenosis is present the pre-systolic murmur associated with this condition disappears and a diastolic murmur related to the second sound remains. This murmur is heard at the apex and Mackenzie maintains that it is diagnostic of auricular fibrillation.

Tracings of the venous pulse in auricular fibrillation reveal an absence of the a wave which in the normal tracing is evidence of auricular contraction.

The electrocardiogram shows the normal ventricular complex with an absence of the auricular or P wave. The rhythm is invariably irregular and and the record between the ventricular complexes is frequently oscillatory.

Forty-five cases of our series, or 61% were markedly decompensated on entrance, and the remainder showed lesser degrees of cardiac embarresment. All cases showed the typical irregularity of the pulse and pulse deficit. In the majority of cases the heart was enlarged both right and left, and to the left in all cases, these observations being checked in most cases by the x-ray.

Fifty-five per cent of the cases presented evidence of mitral disease. Lewis, in his series found 52%. The diagnosis was clinched in all cases by electrocardiographic records.

The blood picture in no case was distinctive. Positive blood Wassermanns were found in only 7%. Examination of the urine showed a rather high incidence of associated nephritis. Thyroid group 63%, rheumatic group 46%, and fibrotic group 60%.

Infection in the teeth and jaws was present in 80%.

Prognosis

The prognosis of auricular fibrillation is not favorable. The condition when once present is usually finally permanent.

Mackenzie and others record occasional cases of this condition in which the patient enjoyed normal pursuits for ten and even twenty years. In these hearts there was no history or clinical evidence of myocardial damage.

Another type of case is one in which the fibrillation is paroxysmal and is precipitated by overexertion, excessive emotion, poisons, etc. Even

here, the prognosis is not good, and the paroxysms occur with increasing frequency and the condition tends to become permanent.

Of the three main groups of cases with which we have mainly concerned ourselves, the thyroid group offers the best prognosis if surgical interference is initiated soon after the onset of fibrillation. Anderson[13] states that auricular fibrillation of recent onset usually disappears on thyroidectomy. Indeed in the light of modern thyroid surgery, it would seem almost criminal to allow a case of hyperthyroidism to develop auricular fibrillation.

The rheumatic and fibrotic group of cases present the most grave prognosis. In both these groups there is present a greater or less degree of myocardial pathology and even under treatment the end usually supervenes in from one to three years.

The condition of the myocardium, then, as indicated by the response to effort is the prime factor in determining the prognosis. The response to treatment is also a point of prognostic importance.

Rarely sudden death occurs in patients with

fibrillation. These deaths are attributed either to fibrillation of the ventricle or to the lodgement of detatched intramural clots in the brain.

In our series as a whole, twenty-two patients or 30% died in the hospital. Of these, five were from the thyroid group, a group mortality of 63%. This should emphasize the importance of early thyroidectomy before cardiac complications occur.

Six deaths occurred in the hospital among the rheumatic group or 16%. Two more patients died within two months after their dismissal from the hospital.

The hospital mortality in the fibrotic group was 40%. One patient died three months after dismissal and another after two years.

These figures as to final outcome are admittedly meagre, but were all that could be obtained from hospital and dispensary records. One must take into consideration that the class of people represented are not usually hospitalized until the disease condition is rather far advanced.

Treatment

The rationale of the treatment of auricular fibrillation is myocardial conservation. Obviously ventricular exaustion as a result of inefficient, disordered action may be prevented in two ways. Either the number of impulses gaining access to the ventricle via the av node may be reduced, or the generation of such impulses inhibited with the restoration of the sinus rhythm. Digitalis accomplishes the first-named effect and quinidine the second.

Digitalis has long been used empirically in the treatment of heart failure. It was long ago included in the pharmacopeia of Wurttemberg but, in 1721 only in that of London, and was soon dropped from the latter because of its so-called danger. However in 1785, William Withering pointed out its marvellous effects in cardiac dropsy, and since that time, digitalis has been the drug of choice for the treatment of heart failure in general and auricular fibrillation in particular.

Digitalis is obtained from an herbaceous plant Digitalis purpurea. The leaves, the flowers, and the seeds present an increasing richness in active principles, but the lamina of the leaf

the medicinal power of which is almost constant, is alone used in therapeutics. The leaves of the second year's growth are gathered just as the plant is beginning to flower, deprived of their midribs, and dried at a low temperature. For uniformity the drug is standardized physiologically.

Of great practical importance is the fact that in auricular fibrillation, more than in any other condition, digitalis slows the heart rate. This slowing is due, according to the most widely accepted theory, to an increased block between the auricles and ventricles which reduces the number of impulses affecting the ventricular myocardium. Thus, when the conductivity of the bundle is depressed by digitalis, the ventricles beat more slowly and often more regularly, even though abnormal auricular activity continues. The drug is also presumed to increase the tonicity of the myocardium and make for stronger contraction. Thus, with a slower and more regular ventricular action, each ventricular contraction becomes mechanically effective and the pulse deficit tends to disappear. This is the most important cause of the improved circulation which usually follows the administration of digitalis to patients suffering from auricular fib-

rillation. The apparent diuretic effect of digitalis is an accessory property of the drug possessed by virtue of the improved circulation.

Digitalis has a cumulative effect and is toxic if therapeutic limits are over-stepped. Nausea and vomiting, headache, and slowing of the pulse indicate that the therapeutic effect has been obtained.

Many preparations of digitalis are and have been used. However for all practical purposes it seems to be generally admitted that none are superior to the tincture or the dried leaf. The latter may be prescribed in pill or capsule form.

The clinical indication for the type of dosage is the degree of circulatory impairment. Rapid digitalization is only indicated when time is the essential factor, as in cases with dyspnea to the point of distress or with severe anasarca. In the majority of cases administration may be conveniently spread over several days or a week.

The work of Eggleston demonstrated the relation of body-weight to dosage of digitalis. The amount of the drug which must be administered in order to obtain full therapeutic effect is roughly directly proportional to the body-weight. The required dose is equivalent to one cat unit per ten

pounds of body weight. One cat unit is equivalent to the activity of .1 gram or 1.5 grains of the dried leaf or 1cc or 15 minims of the tincture.

Hence it is possible to obtain the therapeutic effect of digitalis in several hours by giving at one time the total calculated dose of the drug. In practice, to guard against untoward results in cases of unusual susceptibility, it is customary to divide the dose. Thus in urgent cases when it is certain that the patient has received no member of the digitalis group within fourteen days, one-half the total calculated dose may be given as an initial dose, one-fourth after six hours, and one-sixth after a further six hours. In absence of the onset of therapeutic effect one-tenth of the total may be given every six hours thereafter until signs of therapeutic effect appear.

In non-urgent cases the total calculated dose may be divided so as to secure digitalization in any convenient number of days. In this case the amount of the drug eliminated from the body every day must be allowed for. Experiment has shown that the elimination on the average is equivalent ot about two grains of the dried leaf or 25 minims of the tincture daily.

Crummer[18] favors the block method of administration which he states stands midway between the single massive dose and the chronic administration of the drug. One gram of clinically proven powdered leaves divided into eight doses are given in the course of four days. When no favorable results follow the administration of the first round, it is perfectly safe to double the dosage during the second round, but scarcely ever will there be any favorable results from this increased dosage. He also recommends that an interval of four days be maintained between blocks of digitalis stating that subsequent administration for a similar period will bring about added improvement. Some clinicians prefer to continue digitalis in doses sufficient to maintain digitalization after heart failure is relieved. Crummer reserves digitalis for the time when decompensation occurs.

The improvement in the patient with auricular fibrillation under digitalis therapy is usually striking and immediate. The urinary output increases, edema diminishes, the dyspnea is relieved, and the deficit reduced. The heart rate should be maintained at 90 or slightly less. Rates much lower are frequently manifestations of poisoning rather

than benefit from digitalis.

Failures in the treatment of auricular fibrillation with a potent preparation of digitalis in sufficient dosage are not due to improper selection or use of the drug, but to heart muscle which will not respond, and there always comes a time in the progress of heart muscle disease, when no effect can be obtained from digitalis.

There are certain adjuvants to digitalis medication. Rest in bed should always be enjoined during digitalization. When orthopnea is present the administration of morphine at night is essential. Its constipating effect should be counteracted with a cathartic the following morning. Crummer recommends senna tea for this purpose. When passive congestion of the liver is outstanding, mercury and chalk in three to five grain doses at night followed in the morning with a saline cathartic twice a week, is beneficial. Diuretin given in the interval between blocks of digitalis continues the favorable effect of the drug, especially in increased urinary output, and also dedigitalizes the heart muscle and makes the subsequent administration of digitalis mor effective.

An additional point in the administration of

digitalis remains to be mentioned. Digitalis therapy is not infrequently unsuccessful in the presence of achlorhydria. When such a condition if found to exist, dilute hydrochloric acid should be administered with the drug.

Quinidine is the drug of choice for the treatment of auricular fibrillation per se. Ever since the discovery of quinine alkaloid in 1820 by Pelletier and Caventou it was recognized that quine exerts a very distinct action on the heart. From 1890 onward, in various experiments on the perfused heart it was found to have a sedative influence on the functions of the myocardium, and to diminish excitability and conductivity. In a general way it was found to have a similar effect on the nerves. Wenckebach used quinine in auricular fibrillation and other forms of arrhythmia but came to no formal conclusion. In 1917, Hecht reported cases of complete arrhythmia and of extrasystoles favorably influenced by the intravenous injection of quinine. Frey in 1918 proposed to replace quinine by quinidine which he had found to have a remarkable elective action in regulating the cardiac rhythm.[19]

Unlike the effect of digitalis, which acts by lessening the conductivity of the His bundle, and

not by reestablishing normal sinus rhythm in the auricles, the effect of quinidine is to reestablish normal auricular rhythm. Lewis and his collaborators found that the drug depressed the vagus and at the same time acted directly upon the heart muscle to slow conduction and to lengthen the refractory period. According to the circus contraction theory of fibrillation, the quinidine stops fibrillation through prolonging the refractory period. Hence, the drug establishes a barrier that the circus contraction cannot pass; the circulating wave finds itself blocked and stops short. Under these circumstances, the sinus rhythm reappears, the transition from one rhythm to the other necessarily being abrupt, as may be observed in practice; but, since quinidine acts only on the disordered rhythm without having any influence on its cause, we should expect that the arrhythmia would reappear as soon as the organism is free from the influence of the drug. This expectation, itoo, is borne out in practice. In almost all cases the arrhythmia reappears after several months, or weeks, or even a few days, the patient then becoming refractory to the drug.

The restoration of normal rhythmis usually ac-

companied by an improvement in subjective symptoms and a greatly increased range of activity.

The results of quinidine therapy will differ according to whether the arrhythmia is uncomplicated or associated with cardiac lesions. In the uncomplicated case, the results are impressive, even marvellous, the patient suddenly becoming aware that his heart has resumed its regularity after two or three days use of the remedy. The results are markedly better in otherwise normal hearts when the drug is used within a month of the onset of fibrillation--otherwise the length of the duration of the condition seems to be of little import.[20] The onset of fibrillation in a patient enjoying good or moderate health is a medical emergency and quinidine should be used.[21]

Quinidine is the best treatment for paroxysmal fibrillation whenever the attacks are of such length and frequency as to justify continued medication provided the patient is well enough in the intervals.[21]

The results of quinidine therapy in rheumatic hearts with valvular lesions are less encouraging, although the treatment is more likely to be successful than in the fibrotic group. In general,

cases which have never been decompensated, and especially the cases in which the irregularity is not of long duration will respond in greater proportion. From this standpoint, cases of mitral stenosis must be carefully watched and fibrillation treated at the first onset.[18] Levine and Wilmaers go so far as to say that the drug is of no practical value in the treatment of auricular fibrillation in patients who have previously had congestive heart failure.[22]

The quinidine treatment is not without danger, and in every case the possibility of obtaining results must be carefully balanced against possible harm from its use. In addition to producing much temporary effects as nausea, headache, tinitis, depression, palpitation and tachycardia, quinidine may for a time increase decompensation and even cause sudden death during or shortly after the period of administration. In most cases death is due to cerebral embolism as the result of the liberation of intrauricular clots with the resumption of normal auricular contractions. In some cases, however, the contractile power of the heart muscle is so depressed that it becomes ineffective, due in all probability to the toxic effect of the drug on the myocardium. The drug is definitely contraind-

icated in patients who have shown signs of embolism.

In cases of decompensation associated with fibrillation authorities agree that the decompensation should first be treated with digitalis.[18,19,20]

It is best to administer one or two 3 grain doses to rule out quinidine idiosyncrasy. Then the drug may be administered in increasing doses--10 grains the first day, 15 grains the second, etc. Usually the normal rhythm returns when the patient has ingested 45 to 60 grains. If there is no effect by the sixth day, there is no use in continuing the medication. When the normal rhythm is established it is advisable to continue small doses of quinidine for weeks or even months for the purpose of preventing the recurrence of the irregularity.

In our series all patients but three were decompensated on entrance, and hence received digitalis. In all but 15 improvement, usually marked, was manifested after administration of the drug. The majority of the patients also received diuretin in accordance with the scheme stated previously.

Quinidine alone, was used in only one case--fibrillation with toxic adenoma. Regularity was restored but the patient died suddenly, presumably from cerebral embolism. An autopsy was not done.

Quinidine was employed after decompensation had been treated with digitalis in nineteen cases. In five cases fibrillation was absent on dismissal from the hospital. Follow-up information on these five cases was not available.

Digitalis without doubt is the most valuable drug in the treatment of auricular fibrillation whereas quinidine is of value only in carefully selected cases.

In Conclusion

Although considerable light has been thrown on auricular fibrillation during the last few years of the nineteenth century and these first few years of the twentieth, we are, to say the least, still in semi-darkness concerning this condition.

Until we know the cause or causes of auricular fibrillation we can but treat palliatively, not curatively.

The finger of contemporary investigation points to the finer circulation of the heart and to the nervous system as a possible key to the riddle. Undoubtedly study along these lines will throw more light on the subject of cardiovascular pathology in general and auricular fibrillation in particular.

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