Individual Study

A Study of Auricular Fibrillation in a Country Practice

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AURICULAR FIBRILLATION is a common disorder of cardiac rhythm. In the majority of cases it can be recognised by simple clinical examination at the bedside or in the consulting-room without recourse to elaborate instrumental aids, and, by a judicious selection of therapeutic weapons it is possible, for a variable time, to hold off the final cardiac defeat. These considerations suggested auricular fibrillation as an appropriate study for a family doctor working in a country district. It was therefore decided, in 1947, to begin this study and to continue it for five years. The results are here presented.

(1) *Frequency.* During the five-year period twenty-five patients suffering from auricular fibrillation have been examined and their response to treatment studied. This figure does not include instances of auricular fibrillation occurring as an agonal phenomenon nor does it include any patient who, for any reason, could not be kept under continuous observation for at least one month. Owing to the incidence of certain local factors the size of the practice-population from whom these patients were recruited has varied from year to year but, in round figures, it has averaged about 1,500.

(2) *Sex Incidence.* The numbers in each sex were almost equal viz. eleven males and fourteen females.

(3) *Age Incidence.* The ages of the patients when auricular fibrillation was first detected ranged from 44 years to 97 years: this does not necessarily correspond to the ages at first onset of fibrillation which, especially in the paroxysmal cases, cannot be accurately ascertained.

In ten year periods, the age and sex incidence was as follows :—

Age group	41-50	years	...	...	1 female
,,	,,	51-60	,,	...	...	5 females, 1 male
,,	,,	61-70	,,	...	...	3 females, 6 males
,,	,,	71-80	,,	...	...	1 female, 1 male
,,	,,	81-90	,,	...	...	3 females, 3 males
,,	,,	91-100	,,	...	...	1 female

(4) *Etiology*. Three broad groups of cases could be recognised in this study :—

A. Non-cardiovascular—one case. B. Cardiovascular—21 cases.

C. Unknown—three cases.

Group B. cases fell into three sub-groups.

(i) Valvular heart disease—(a) rheumatic—three cases—all females; (b) probably non-rheumatic—two cases—both females.

(ii) Hypertensive heart disease—12 cases—five males, seven females. All the patients in this sub-group had diastolic blood-pressure readings consistently above 90 mm. Hg. on repeated unhurried examinations in the supine position.

(iii) Ischaemic heart disease—four cases, all males.

No example of auricular fibrillation due to thyrotoxicosis was discovered during the course of this study.

The non-cardiovascular case occurred in a woman of 68 years, who in the course of an irregularly febrile illness characterised by normocytic, normochromic anaemia, leukopenia, purpura and obscure chest-signs, developed a paroxysm of auricular fibrillation lasting 24 hours, with spontaneous reversion to normal sinus rhythm. She was shortly thereafter admitted to the Royal Infirmary, Perth, and died there five weeks later. An ante-mortem diagnosis of acute aleukaemic monocytic leukaemia was made in hospital.

Both examples of auricular fibrillation in patients with probable non-rheumatic valvular heart disease occurred in elderly women aged 88 years and 86 years respectively. In the first of these the clinical diagnosis is aortic stenosis despite the admitted rarity of this lesion as a cause of auricular fibrillation. The age and general condition of the patient precluded a journey of 27 miles in order to examine the heart, and especially the aortic valve, radiologically. The second patient had a constant and well-marked double aortic murmur. It is suggested that in both cases the underlying lesion is sclerosis of the aortic valve.

Three patients are included in the group labelled " unknown etiology." The first of these, a man of 69 years, developed a short paroxysm of auricular fibrillation while apparently in normal health. The attack lasted for only a day and a half and reverted to normal sinus rhythm with a total dosage of only 2.5 mgm. digoxin given orally. The blood pressure prior to the onset of the arrhythmia varied between 150-170/80-84 m.m. Hg. Comparable levels were not reached until above five weeks after the episode. There

was no pain, no pyrexia and no sign of cardiac failure. About four to five days following the attack an Evans-type third heart sound was heard. Three standard leads and chest leads C2R and *IV* R revealed no E.C.G. evidence of heart disease. It is probable that this patient had in fact a small myocardial infarct which unipolar leads—not then available for this study—might have disclosed. This single episode took place nearly eighteen months ago and there has been no known recurrence. The second patient is a man of 65 years who suffers from generalised arterio-sclerosis : the symptoms are mainly cerebral and consist of giddiness on exertion, sleeplessness, and a Parkinsonian type of tremor without rigidity. Blood pressure lies within the zone 110-120/70-80 m.m. Hg. The fibrillation is permanent. The third patient was a very old lady who had her first known paroxysm of fibrillation at the age of 96 years : she had two further known paroxysms, both of short duration, in the ensuing three years and she ultimately died at the age of 99 years and 8 months in normal sinus rhythm. All three episodes of arrhythmia were converted to normal sinus rhythm with short courses of oral digoxin the total dosages varying between 1.0 mgm. and 2.25 mgm. There was no diastolic hypertension and no sign of either right-sided or left-sided heart failure. It is a reasonable surmise that this very old lady fibrillated because she was a very old lady.

(5) *Precipitating factors.* An attempt was made to establish the precipitating factor or factors in all cases, but without success. In 17 instances the precipitating factor is unknown: in three instances the onset of the arrhythmia was immediately ante-dated by an attack of upper respiratory catarrh: two patients attributed their attacks to recent undue physical exertion ; in one instance there was recent physical exhaustion combined with excess alcohol, one episode occurred in the course of a recent anterior myocardial infarct and one was incidental in the course of acute aleukaemic monocytic leukaemia.

(6) *Presenting symptom of the onset of fibrillation.* An attempt was also made to separate out the symptomatology of the onset of auricular fibrillation from that of the underlying disease—if any. There were only 15 occasions on which this could be done with reasonable confidence:—dyspnoea was the presenting symptom in six instances, " palpitation " in four, dyspnoea and " palpitation " in one, " collapse " in one, and retro-sternal discomfort or pain in three.

(7) *Mortality.* Eight of the 25 patients studied over the past five years have died. Their sex, ages at death, type of fibrillation underlying disease and causes of death are shewn in Table 1.

TABLE 1

Sex	*Age at Death in years*	*Type of Fibrillation*	*Underlying disease*	*Cause of Death*	*Remarks*
F	89	Permanent	Sclerosis of aortic valve	Cardiac defeat	Well marked double aortic murmur with L.V. failure.
F	54	Permanent	Rheumatic heart disease	Cardiac defeat	Classical mitral stenosis, estimated total duration 17 years.
F	99.2/3	Paroxysmal	Nil found	Extreme age	
F	68	Paroxysmal	Acute aleukaemic monocytic leukaemia		
F	84	Permanent	Hypertensive heart disease	Cardiac defeat	
F	72	Paroxysmal	Hypertensive heart disease	? Cardio-vascular catastrophe	Found dead in bed. No P.M.
M	68	Paroxysmal	Ischaemic heart disease	? Acute coronary occlusion	Died suddenly 48 hours after minor operation.
M	85	Permanent	Hypertensive heart disease	Acute coronary occlusion	

(8) *E.C.G. investigations.* These were carried out on nine of the 25 patients, using three standard limb leads and up to six bipolar chest leads : (the indifferent electrode for the latter is on the right arm in the electro-cardiograph used). This small investigation resulted in one patient, a man who had recently recovered from a short paroxysm of fibrillation, being transferred from the hypertensive to the ischaemic sub-group. In the other eight patients no additional information was obtained wherewith to supplement the bedside findings. Admittedly the investigation was technically inadequate by modern standards in that unipolar limb leads and unipolar chest leads were not used.

I am indebted to Dr. Rae Gilchrist of Edinburgh for the elucidation of a further case—that of an introspective over-anxious man of 65 years whose vague feelings of retro-sternal numbness, not precisely related to exertion, were found after standard exercise, to be associated with short bouts of paroxysmal fibrillation with E.C.G. evidence of coronary insufficiency.

Treatment

(i) *General Treatment* was on accepted lines—bed-rest where indicated, with " freedom of the bed " as soon as possible, followed by instructions regarding diet, exercise and planned rest-periods by day and at week-ends.

(ii) *Drug Treatment.* Digitalis was the main weapon, in the form of Tab. Digoxin B.P. 0.25 mgm. ; intravenous digoxin was not needed in any patient. Quinidine sulphate orally was used in a small number of suitable cases ; aminophyllin, orally, intravenously or rectally; mersalyl by intramuscular injection; hypnotics and sedatives, were exhibited where indicated. A salt-poor diet was prescribed for those patients in whom mersalyl was not being regularly employed.

(a) *Digoxin treatment.* (i) INITIAL DOSAGE. Except in the very elderly, the initial dose of digoxin given was 1.0 mgm. (four tablets) followed by 0.5 mgm. every six hours until either a resting apex rate of 80 per minute or under was attained, or signs and symptoms of digitalis intoxication supervened.

Over the five year period 20 patients were treated with digoxin in the above dosage or minor variations thereof. In 11 of these patients the basic arrhythmia was unaltered and they are considered to be examples of permanent fibrillation. Of these 11 patients five were adequately controlled and six were not adequately controlled, either because of poor co-operation or because of the incidence of signs and symptoms of digitalis intoxication. The remaining nine patients were examples of temporary fibrillation and they had between them a total of 13 episodes of fibrillation all of which were converted to normal sinus rhythm with digoxin alone. Of these nine patients one has died (of extreme old age and in normal sinus rhythm up to the time of death), one has relapsed to permanent fibrillation and the remaining seven have remained in normal sinus rhythm for periods varying from two months to four and a half years at the time of writing. The total amount of digoxin initially required in the successfully treated episodes varied from a minimum of 1.0 mgm. to a maximum of 5.25 mgm.

Signs or symptoms of overdosage occurred on nine occasions: these cleared without ill-effects on stopping digoxin completely. The total amount of digoxin required to produce symptoms of intoxication varied from a minimum of 1.0 mgm. to a maximum of 4.75 mgm. No relationship could be observed between the apex-rate at the onset of treatment and the subsequent development of digitalis intoxication. In a very general way, the very elderly were found to tolerate digoxin less satisfactorily than the younger-age-groups irrespective of dosage—there were six instances of digitalis

intoxication at ages 70 and over, compared with three instances in the younger age groups.

(ii) MAINTENANCE DOSAGE. The onset of symptoms of digitalis intoxication has been a limiting factor in maintenance dosage as in initial dosage : e.g. in one patient a total weekly maintenance dosage as low as 1.5 mgm. digoxin induced such symptoms. Within the limits set by this factor the optimum attainable weekly dosage varied between 1.5 mgm. and 4.5 mgm. It is well known that reasonably intelligent patients can, after a longer or shorter period of trial and error, regulate their own maintenance dosage with reasonable efficiency and this plan has been followed in the present investigation.

(b) *Quinidine treatment.* Oral quinidine sulphate either with or without a preceding course of digoxin was employed in six episodes of auricular fibrillation occurring in five patients: three of the patients had hypertensive heart disease, one had ischaemic heart disease and one had rheumatic valvular heart disease. The criteria for the employment of quinidine were those commonly accepted viz. auricular fibrillation of recent onset without evidence of gross cardiac pathology. An exception was made in the case of the last-named patient, a woman of 52 in whom fibrillation appeared to have been present for not more than a year before she came under care, but in whom the signs of mitral stenosis were very obvious. Her apex-rate could not be controlled by digoxin before symptoms of intoxication set in and as her livelihood depended on her being able to resume work within the near future it was decided, after seeking a second opinion, that quinidine therapy was a justifiable risk. Fortunately she reverted to normal sinus rhythm without mishap and she has remained in that rhythm for five months at the time of writing.

All the patients were in bed while undergoing quinidine therapy and the dosage schedule used was that recommended by White (1944) :—After a test dose of 0.2G to exclude idiosyncrasy to the drug, 0.4G was given every two hours for five doses on the first day of treatment, continuing if necessary with a similar course on the second day. Reversion to normal sinus rhythm was secured in all cases : the total amount of quinidine necessary to attain this end varied from a minimum of 2.0G to a maximum of 4.2G and the remissions lasted from a minimum of one month to a maximum of two and a half years. Happily there were no accidents and the only symptom of toxicity was temporary tinnitus in the higher range of dosage. Of the five patients treated, one has died, of acute coronary occlusion 48 hours after undergoing a minor operation, and four are alive. Of these one patient has had three further episodes of fibrillation which have on each occasion been converted

to normal sinus rhythm with digoxin, one patient has relapsed to permanent fibrillation and the remaining two have remained in normal sinus rhythm for five months and 16 months respectively at the time of writing.

To the student of cardiac therapeutics, there can be few experiences more satisfying than the conversion of the delirium of auricular fibrillation, to the steady beat of normal sinus rhythm under the influence of quinidine, and the urge to extend the use of this drug more widely proved at times almost irresistable. It was, however, realised that the constant observation and care required of patients undergoing quinidine therapy, including if possible E.C.G. control, is seldom practicable in general practice, and it was decided, albeit with reluctance, to subscribe to the doctrine that " it (i.e. quinidine) is a drug that for the treatment of auricular fibrillation has little place in general practice." (Hill, 1949.)

Discussion. In the course of this small investigation several items of interest came to light. The first of these was the infrequency of rheumatic heart disease as a cause of auricular fibrillation in the patients studied—it could be diagnosed with reasonable confidence in only three patients out of the total 25. This is due to the fact that, although no selection whatever was made of the patients for the study, provided each could be kept under observation for at least one month, they all fell into the middle age and old age groups. It may well be that, with the gradual reduction in the incidence of rheumatic heart disease in children and young adults together with the relatively rapid ageing of the population generally, this disease may soon be displaced from its long held primacy as the commonest cause of auricular fibrillation in favour of the degenerative forms of heart disease, the hypertensive and the ischaemic. During the course of this study other types of cardiac arrhythmia have been looked for. With the exception of the almost universal ventricular extra-systole, auricular fibrillation was the commonest arrhythmia found. The second item of interest was the relative frequency with which auricular fibrillation was converted to normal sinus rhythm by digitalis. Of the 20 patients treated with digoxin over the past five years, seven have remained in normal sinus rhythm for periods varying from two months to four and a half years at the time of writing. Obviously the question arises, whether a similar reversion might not have occurred by merely putting the patient to bed without the administration of a ' cardiotonic ' drug. This certainly happened in the case of the patient suffering from aleukaemic leukaemia noted above and also, almost by accident, in another patient in this study. She was an over-weight, hypertensive woman whose apparently permanent

fibrillation was being adequately controlled by digoxin. She developed thirst, polyuria and glycosuria and while in the local hospital for the investigation and treatment of this additional complication digoxin was stopped: at about the time of the abolition of her glycosuria, by dietetic measures only, she reverted to normal sinus rhythm and she has remained in that rhythm without digoxin for 15 months at the time of writing. Her urine remains sugar-free: she remains hypertensive. On the other hand, a patient not included in this study, an over-anxious hypertensive man in his sixties, who very recently developed his first known paroxysm of auricular fibrillation, was kept in bed on sedatives only for one week without any effect on his arrhythmia. On the exhibition of digoxin he reverted to normal sinus rhythm.

There can be few drugs in the pharmacopoeia which have been so closely studied alike by the experimentalist and by the clinician as the drugs of the digitalis group ; but the precise mode of action of these drugs remains to be elucidated. McMichael (1950) speaks of "our fundamental ignorance of the mode of digitalis action." The same statement might be made about the mechanism of auricular fibrillation since the recent work of Prinzmetal *et al.* (1950) seems to have rendered untenable the hitherto accepted circus-movement theory postulated by Lewis many years ago.

Results

17 of the 25 patients comprising this study are alive: all are ambulant, at ages ranging from 45 years to 89 years. Clinically five patients shew signs of cardiac failure, 12 are free of such signs: of the former, four are in permanent fibrillation.

Summary

1. A report is presented of a study of auricular fibrillation in a country practice over a five-year period.

2. Notes on treatment and the responses of the individual patients thereto are given and the fate of the patients in the group described.

3. Items of interest that occurred in the course of the investigation are discussed. These include (a) the relatively minor role played by rheumatic heart disease in the etiology of auricular fibrillation in this particular study: (b) the reversion of auricular fibrillation to normal sinus rhythm under the influence of digitalis in seven out of 20 patients so treated.

REFERENCES

Hill, I. G. W. Article in *Text Book of Medical Treatment.* Dunlop, Davidson and McNee, 5th Edition, Edinburgh, 1949, p. 665.
McMichael, J. *Pharmacology of the Failing Human Heart.* Oxford, 1950, p. 52.
Prinzmetal *et al. Mechanism of the Auricular Arrhythmias.* Circulation, 1950, **1**, 241.
White, P. D. *Heart Disease.* 3rd Edition, New York, 1944, pp. 895-6.